

# PROCEEDINGS

## VOLUME XLIV

### No. 2

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Physics. — *La pression du toit sur le charbon près du front dans les exploitations par tailles chassantes. Troisième Chapitre. I.* Par F. K. TH. VAN ITERSON.

(Communicated at the meeting of January 25, 1941.)

### TROISIÈME CHAPITRE.

**La pression du terrain autour des vides créés, après la rupture des roches.**

#### § 1. *Introduction.*

Dans deux études préalables <sup>1)</sup> nous avons admis que le rocher, toit et mur <sup>2)</sup>, consiste en matière solide, résistante aux contraintes, dues à la surcharge du terrain. Nous avons démontré que ces contraintes sont concentrées aux entours du vide créé par l'exploitation et qu'aux profondeurs où nous travaillons les tensions calculées selon les lois de l'élasticité surpassent de beaucoup la résistance de la roche, schiste ou grès, qui enserme le charbon. Il est donc prouvé mathématiquement que par l'abatage du charbon la roche environnante se casse. Même quand on admet que le charbon en veine doit être considéré comme de la matière plastique ou friable il est certain qu'à peu de distance derrière le front du charbon la pression s'accroît à des valeurs excessives, qui surpassent la résistance à la rupture du toit et du mur.

Mais nous n'avons pas besoin de calculs compliqués pour arriver à ce résultat. Les observations faites dans les mines le prouvent. Dans son rapport de 1938 l'inspecteur général des mines en Angleterre <sup>3)</sup> a spécialement attiré l'attention sur le grand nombre d'accidents, signalés par presque tous les inspecteurs divisionnaires, dus à la chute de terres dans les exploitations par tailles chassantes. En général ces chutes sont causées par des cassures invisibles du toit, qui se produisent en avant du front de taille. L'examen spécial, ainsi que l'expérience du personnel, ont démontré que ces fissures, généralement cachées à notre vue et presque imperceptibles dans le premier stade de leur développement, naissent parallèlement à la ligne du front à des intervalles réguliers.

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<sup>1)</sup> La pression du toit sur le charbon près du front dans les exploitations par tailles chassantes. Premier et deuxième chapitre. Proceedings Vol. XLII, No. 2, 1939 et Vol. XLIII, No. 2, 3 et 4, 1940.

<sup>2)</sup> Par mur le mineur entend la surface du rocher en dessous de la couche de charbon.

<sup>3)</sup> 18th Annual Report of the Secretary for Mines for the year ended December 1938, and the 31st Annual Report of H. M. Chief Inspector of Mines for the same period.





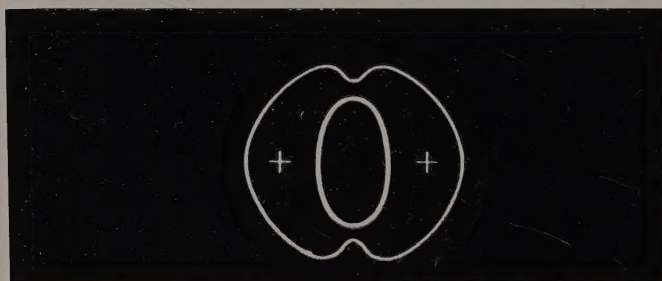


Fig. 4. Examen photoélastique de la répartition des tensions autour d'un trou ou d'une galerie à section elliptique (proportion des axes 2 : 1) et représentation des tensions tangentielles. (Emprunté à „Archiv für bergbauliche Forschung“, Verlag Glückauf G. m. b. H., Essen, Jahrgang 1, Heft 1, Seite 18, Abb. 22, „Belastung 500 kg; Höchstzahl der Streifen 8,0; Streifenzahl für den ungestörten Teil 3,7; Spannungserhöhung  $8,0 : 3,7 = 2,16$  fach, berichtigt auf 2,06 fach.)

Récemment on a appliqué la méthode optique pour déterminer la répartition des pressions autour des cavités minières <sup>1)</sup>.

Nous empruntons quelques figures à l'importante étude expérimentale exécutée à l'aide financière du HELMHOLTZ-Gesellschaft.

D'abord (fig. 1) nous reproduisons la figure obtenue par lumière polarisée à travers une plaque de décorit, munie d'un trou trapézoïdal, chargée verticalement. La forte concentration des lignes près des angles rentrants, beaucoup plus accentuée en réalité que ne l'indique la figure, désigne des pressions excessivement élevées à ces endroits.

Il en est de même des nombreux autres essais sur des modèles de sections de galeries et de sections de champs d'abatage faits par l'auteur.

Pour évaluer la valeur de la méthode optique nous reproduisons sur la figure 2 le résultat obtenu par DORSTEWITZ avec la plaque munie d'un trou rond, comprimée verticalement. La solution mathématique due à KIRSCH <sup>2)</sup>, est bien connue. Si la pression verticale est  $p$ , on obtient une pression  $3p$  aux bouts du diamètre horizontal et une tension  $-p$  aux extrémités de l'axe vertical.

Si nous superposons la même pression en sens horizontal on doit obtenir une pression  $2p$  à la périphérie du cercle.

Selon l'expérience on obtient la figure 3; laquelle indique pour la tension

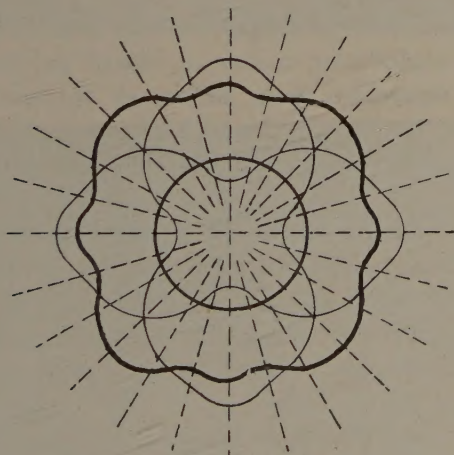


Fig. 3. Superposition d'une pression horizontale et de la même pression verticale, ce qui devait fournir un cercle pour les tensions tangentielles.

tangentielle en moyenne environ 35 % de plus que ne donne le calcul, ce qui est assez satisfaisant pour cette sorte d'expériences.

Sur la figure 4 nous reproduisons le résultat obtenu par le même expérimentateur avec la plaque à trou elliptique, proportion des axes 2 : 1,

<sup>1)</sup> Spannungsoptische Untersuchungen als Beitrag zur Klärung von Gebirgsspannungen um bergmännische Hohlräume, par Dr. Ing. G. DORSTEWITZ. Archiv für bergbauliche Forschung, Aug. 1940, S. 9, 1, Jahrgang, Heft 1.

<sup>2)</sup> Kirsch. Zeitschrift des Vereins deutscher Ingenieure, 1898, S. 805.



chargée verticalement. Le calcul <sup>1)</sup> nous apprend qu'aux bouts du petit axe de l'ellipse les tensions tangentiellees sont des pressions  $2p$  et qu'aux bouts du grand axe, (axe dans le sens de la pression  $p$ ) règnent des tensions tangentiellees  $-p$ , indépendamment de la proportion des axes.

On remarque sur la figure 4, où sont représentées les tensions tangentiellees, que les tensions de traction au sommet et à la partie inférieure du profil n'y apparaissent pas. Or ces tensions, qui doivent se manifester dans tous les profils examinés, sont funestes pour la roche houillère, qui ne résiste pas à la traction.

L'appareillage ne permet que d'appliquer une pression verticale. Pour connaître la répartition des tensions autour du trou quand le modèle est chargé en tous sens, il faut répéter l'essai avec la plaque tournée d'un demi tour et superposer les résultats.

Ces essais mettent en évidence que le rocher doit se casser.

Quand on a une fois attiré l'attention du personnel de mines sur le fait que mise à nu par les travaux souterrains à grande profondeur la roche se relache et se casse, il en trouve la confirmation par d'innombrables observations.

Autour du vide créé par l'exploitation le schiste allégé se dilate, foisonne et les fragments ne s'ajustent plus. Même si les morceaux sont assez grands, ils ne tiennent plus ensemble.

L'échelle du phénomène physique importe peu, il faut considérer le terrain houiller en exploitation comme un massif pulvérulent, composé de grains facilement séparables. Pour mettre en formules ce qui se passe autour de nos chantiers, il faut appliquer les lois pour l'équilibre des massifs à frottement interne qu'on trouve dans les manuels de la mécanique appliquée <sup>2)</sup>.

Les figures 5 et 6, représentant des galeries en cours d'élargissement, expliquent ce que nous entendons par masses incohérentes.

Les plissements géologiques, les essais de VON KÁRMÁN, de BRIDGEMAN et d'autres nous apprennent que les matières pierreuses, soumises à des pressions environnantes assez élevées, se comportent comme de la matière plastique.

En observant les mouvements de la roche vers les vides miniers on était souvent tenté d'y voir une manifestation de la plasticité du schiste qu'on a reconnue dans les plissements géologiques.

Mais nos observations, dont les deux photographies ne donnent qu'une représentation partielle, nous ont prouvé que la soudure des débris par la pression est imparfaite et se distingue peu de celle qu'on constate dans les

<sup>1)</sup> Stresses in a plate due to the presence of cracks and sharp corners by Inglis. Transactions of the Institution of Naval Architects 1913, part I.

Kerbspannungslehre von H. NEUBER, 1937.

Technische Dynamik von BIEZENO und GRAMMEL, 1939.

<sup>2)</sup> Handbuch der Physik. Band VI, 1928, Mechanik der elastischen Körper. Kap. 6. A. NÁDAI: Plastizität und Erddruck V, Das Gleichgewicht lockerer Massen.



F. K. TH. VAN ITERSON: LA PRESSION DU TOIT SUR LE CHARBON PRÈS  
DU FRONT DANS LES EXPLOITATIONS PAR TAILLES CHASSANTES.



Fig. 5 et 6. Par la détente la roche se casse et se comporte comme de la matière pseudo-plastique.





remblais de travaux civils, où l'on admet que la validité des formules de la poussée des terres est parfaitement sanctionnée par l'expérience.

Il ne peut être question de plasticité que quand il y a une pression de quelques milliers d'atmosphères dans tous les sens<sup>1)</sup> et en réalité il ne pèse qu'une atmosphère sur les surfaces mises à nu et dans le sens tangentiel il y a même très souvent des tensions de traction.

Toutes les formules de l'équilibre des masses incohérentes, de la poussée des terres sont donc à notre disposition pour expliquer les mouvements et la pression dans les exploitations souterraines.

## § 2. *L'équilibre des massifs pulvérulents.*

La condition fondamentale qui permet d'exprimer en nombres l'équilibre dans une masse de terre a été formulée par COULOMB, dès la fin du XVIII<sup>e</sup> siècle. Ce physicien admit que l'équilibre résulte du frottement mutuel des grains. Quand on s'imagine une coupe à une place quelconque dans le massif et la matière des deux parties séparées, consolidée, un glissement de l'une partie sur l'autre engendrerait alors une force de frottement. Dans tous les points les contraintes obéiraient à la loi du frottement, la composante normale  $\sigma_n$  étant toujours une compression et la composante de cisaillement  $\tau$  étant dans l'équilibre toujours inférieure à la composante de frottement  $\sigma_n \operatorname{tg} \varrho$ .  $\varrho$  représentant l'angle de frottement. L'équation caractéristique dans un massif pulvérulent est donc

$$-\sigma_n \operatorname{tg} \varrho < \tau < \sigma_n \operatorname{tg} \varrho.$$

Avec les figures 7 et 8, représentant des demi-sphères collées sur une

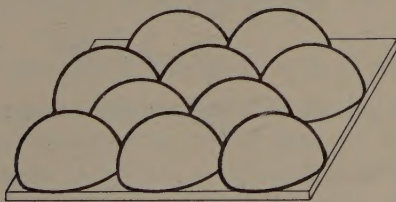


Fig. 7. Modèle pour essais de frottement interne de billes à entassement le plus serré (centres rangés en tétraèdres).

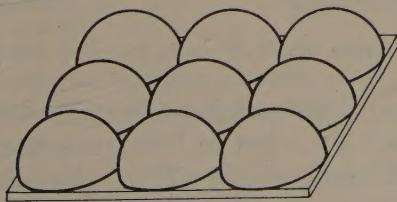


Fig. 8. Modèle pour essais de frottement interne de billes à entassement quadrangulaire.

<sup>1)</sup> DE SITTER, *Geologie en Mijnbouw* 1—1939—(10) p. 225.

planchette, nous tâcherons d'expliquer pourquoi le frottement est proportionnel à la pression. Pour la démonstration il faut se servir de deux planchettes identiques, dont on place l'une en position renversée sur l'autre. L'angle de frottement de schiste sur schiste en repos étant  $32^\circ$ , il suffit d'incliner les planchettes jusqu'à cet angle pour voir glisser la partie supérieure quand on a posé les centres des sphères supérieures en aplomb des centres des sphères de l'autre planchette.

Mais que se passe-t-il quand on a enfoncé les sphères autant que possible dans les vallées mutuelles, quand on a placé les deux planchettes dans la position la plus rapprochée? Alors il faut incliner la disposition d'abord à un angle  $\alpha$  pour faire sortir les sphères supérieures de leur enfoncement sans tenir compte du frottement et puis à un angle  $\beta$  qui tient compte de l'angle de frottement  $\varphi$ .

Le calcul donne pour la figure 7

$$\begin{aligned} \operatorname{tg} \alpha &= \frac{1}{2\sqrt{2}} & \alpha &= 19^\circ 30' \\ \operatorname{tg} \beta &= \frac{2}{\sqrt{3}} \operatorname{tg} \varphi & \beta &= 35^\circ 30' \\ & & \alpha + \beta &= 55^\circ \end{aligned}$$

Pour la figure 8 on obtient:

$$\begin{aligned} \operatorname{tg} \alpha &= \frac{1}{\sqrt{2}} & \alpha &= 35^\circ 30' \\ \operatorname{tg} \beta &= \frac{2}{\sqrt{3}} \operatorname{tg} \varphi & \beta &= 35^\circ 30' \\ & & \alpha + \beta &= 71^\circ \end{aligned}$$

Quand des cubes de schiste sont enchevêtrés comme l'indique la figure 9,

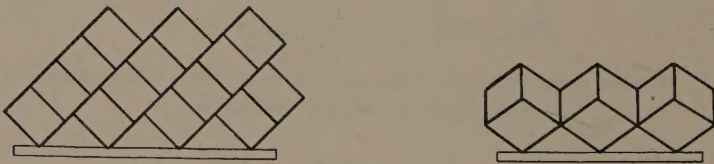


Fig. 9. Cubes rangés et placés sur un de leurs coins.

le glissement se produit si la force normale qu'ils exercent entre eux fait un angle de  $\alpha = 45^\circ$  s'il n'y a pas de frottement et  $\alpha + \beta$  si on ajoute le frottement  $\beta = \varphi = 32^\circ$  donc  $\alpha + \beta = 77^\circ$ . Si les cubes sont mis à plat il n'y a que le frottement sous l'angle  $\varphi = 32^\circ$ . Le glissement de débris de schiste se produit donc selon l'inclinaison du plan de contact sous un angle entre  $32^\circ$  et  $73^\circ$ .

Le chiffre de  $32^\circ$  pour l'angle de frottement entre schiste et schiste a



été déterminé par nous. Le coefficient de frottement est donc  $\operatorname{tg} \varphi = 0,625$  (au départ).

Dans les manuels à l'usage des ingénieurs on trouve pour pierre calcaire sur pierre calcaire des valeurs entre  $\operatorname{tg} \varphi = 0,75$  et  $0,67$  au départ, et entre  $0,67$  et  $0,60$  en mouvement. Pour  $\operatorname{tg} \varphi = 0,67$ ,  $\varphi = 34^\circ$ .

On verra plus tard pourquoi nous tenons beaucoup à connaître le chiffre exact.

CAQUOT<sup>1)</sup> a prouvé que le coefficient de frottement apparent  $\operatorname{tg} \varrho$  est donné par  $\operatorname{tg} \varrho = \frac{\pi}{2} \operatorname{tg} \varphi$  quand les plans tangents aux particules ont une orientation également probable dans toutes les directions. Ceci donne pour  $\varphi = 32^\circ$ ,  $\operatorname{tg} \varrho = \frac{\pi}{2} \times 0,625 = 0,983$ ,  $\varrho = 45^\circ$  ce qui est conforme à la réalité, quand on a entassé le schiste avec un peu de soin.

Il faut remarquer que l'enchevêtrement des morceaux a encore un autre effet que d'augmenter le frottement interne.

L'entassement dans le massif intact est très serré. Les mouvements se produisent seulement par glissements sur les plans tangents au contact des fragments et non pas par un déplacement général suivant une surface qui traverserait tous les débris. La théorie dont nous parlons dans le paragraphe suivant nous apprend que les glissements internes dans la masse mouvante se manifestent toujours selon des surfaces conjuguées infiniment nombreuses. Si l'on pense à un massif composé de morceaux arrondis ceux-ci doivent sortir de leurs encoches. Il en résulte une dislocation, un desserrage de l'entassement dense, un accroissement de volume, une diminution considérable du frottement interne, de l'angle de frottement apparent  $\varrho$  dès que la masse est en mouvement.

§ 3. *L'inclinaison du plan d'affaissement et la pression sur le soutènement.*

Dans l'annexe nous donnons le calcul pour ce cas de la poussée des terres. C'est l'exemple le plus clair de la répartition des pressions dans le terrain en glissement.

Le résultat est (voir figures 10 et 11)

$$\sigma_y = \frac{1 - \sin \varrho}{1 + \sin \varrho} \gamma z \quad \sigma_z = \gamma z \quad \tau = 0 \quad \theta = \frac{\pi}{4} + \frac{\varrho}{2}.$$

Ici  $\varrho$  est l'angle apparent de frottement interne;  $\gamma$  le poids spécifique de la masse meuble. Quelques lignes de glissement sont indiquées dans la partie gauche de la figure 10.

La formule très simple qu'on trouve pour l'angle d'affaissement  $\theta = \frac{\pi}{4} + \frac{\varrho}{2}$  met fin à une controverse entre ingénieurs de mines sur la

<sup>1)</sup> Equilibre des massifs à frottement interne par ALB. CAQUOT, Paris 1934.

question de savoir dans quelle mesure  $\theta$  dépend de la nature du terrain et de l'inclinaison de la couche exploitée.

Dans la table suivante nous donnons les angles  $\varrho$  et  $\theta$  pour quelques espèces de terrain houiller en morceaux et pour les morts terrains.

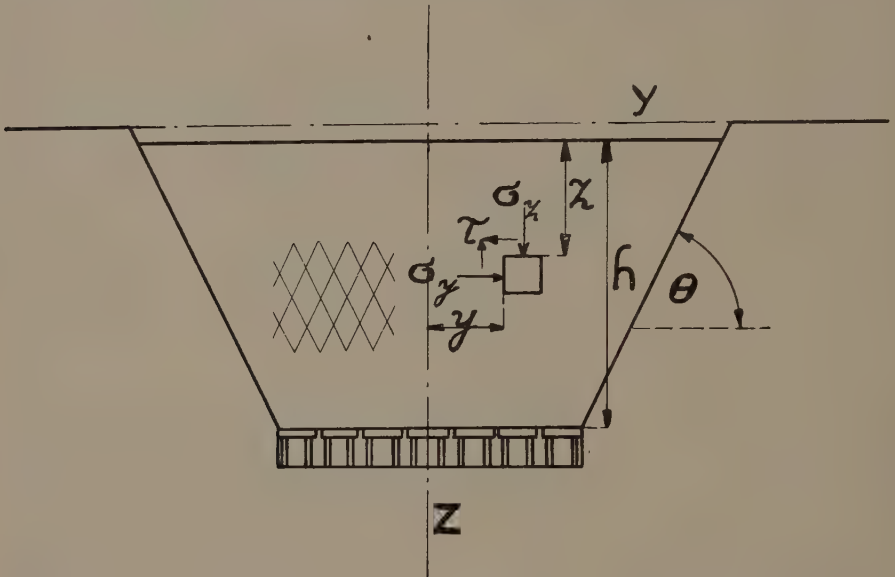


Fig. 10. Angle d'affaissement  $\theta$  dans le cas d'un soutènement compressible.

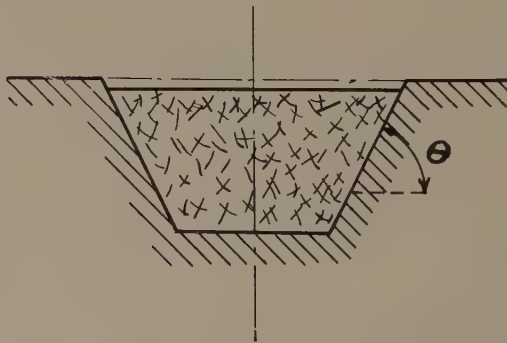


Fig. 11. Masse de terre affaissée dans une exploitation abandonnée.

Nous empruntons  $\varrho$  l'angle du talus = l'angle de frottement interne à HÜTTE, Des Ingenieurs Taschenbuch 26. Auflage 1936 p. 719.

Il ne faut pas perdre de vue que ces valeurs sont des minima et que  $\varrho$  est plus grand quand on place les débris de roche avec quelque soin.

Il est physiquement impossible que les affaissements de la surface, causés par les exploitations minières, se manifestent au delà de cette limite de  $55^\circ$ .

Les angles d'affaissement  $\theta$ , ainsi calculés, doivent être considérés comme les minima. Les angles des talus naturels dont nous les avons dérivés ont été mesurés au remblai de la matière.



Espèce de terrain.	Angle de talus et de frottement interne $\varrho$ en degrés.	Angle d'affaissement par exploitation minière $\theta$ en degrés.
Charbon à gaz sec ou humide	45	$67\frac{1}{2}$
Autres espèces de charbon	30—45	$60-67\frac{1}{2}$
Roche calcaire	30—45	$60-67\frac{1}{2}$
Schiste en gros morceaux, comprimé (p.e. le terril)	38	64
Pierres concassées humides et sous pression	35—40	$62\frac{1}{2}-65$
Sable humide peu serré	27	$58\frac{1}{2}$
Sable sec peu serré	32	61
Terre, sable, gravier sous pression, sec	30—45	$60-67\frac{1}{2}$
Terre remblayée	27—40	$58\frac{1}{2}-65$
Gravier humide sous pression	25	$57\frac{1}{2}$
Terre glaise ou argileuse sèche sous pression	40—46	65—68
La même, humide	20—25	$55-57\frac{1}{2}$

Pour la roche cassée en site où l'enchevêtrement est plus prononcé qu'au remblai de schiste déversé par tombereau, l'angle de frottement apparent  $\varrho$  et par suite l'angle d'affaissement  $\theta$  sont plus grands. Au lieu de  $\varrho = 38^\circ$  pour le schiste, comme le donne la table précédente on aura  $\varrho = 45^\circ$ , le chiffre théorique déduit selon CAQUOT, et davantage pour les morceaux de calcaire ou de grès.

On obtient donc pour l'angle d'affaissement du schiste en site:

$$\theta = \frac{\pi}{4} + \frac{\varrho}{2} = 45^\circ + 22\frac{1}{2}^\circ = 67\frac{1}{2}^\circ.$$

Le géomètre en chef des Mines de l'Etat à Heerlen (Hollande), donne les chiffres suivants <sup>1)</sup> dans sa thèse de docteur:

Ces angles d'affaissement ont été déterminés avec une précision extrême et sont en parfait accord avec l'inclinaison de  $67^\circ 30'$  calculée par nous selon la théorie de l'équilibre dans les massifs à frottement interne.

Y a-t il une meilleure preuve possible que le terrain houiller, au dessus

<sup>1)</sup> Gebirgsbewegungen bei Steinkohlenbergbau von G. J. A. GROND, Inaugural-Dissertation. Geologisches Institut der Westfälischen Wilhelms-Universität zu Münster, 1926, p. 30.

Observation No.	Inclinaison de la couche de charbon:	Inclinaison du plan d'affaissement:
1	— 8°	68°
2	+ 7°	68° 45'
3	+ 5°	68° 20'
6	— 8.5°	68° 20'
7	— 3°	68° 40'
8	—10°	67° 10'
10	—22°	67° 20'
11	—15°	67° 15'
14	— 9°	65° 40'

des champs d'exploitation, consiste en un amas de débris de pierres enchevêtrés?

Quand l'enchevêtrement diminue, quand les débris de roche se disloquent, quand la masse est en mouvement et que de ce chef le coefficient de frottement dans les surfaces de contact des morceaux tombe, l'angle de frottement décroît pour le schiste de 45° à 36° environ et par conséquent les plans d'affaissement deviennent moins escarpés,  $\theta$  diminue de 67° 30' à 63°. Les plans d'affaissement s'inclinent davantage et parcourent une zone de transition. Il est facile d'expliquer qu'au dessus du chantier d'exploitation le terrain est comprimé dans le sens horizontal et étiré dans le sens vertical, tandis que dans la zone de transition la surface de la terre s'allonge.

Quand les morts terrains et le schiste sont humides, ce qui est très souvent le cas, il y a un effet de temps très prononcé. L'eau ne se déplace que lentement dans les pores des terres fines et comprimées. Par les frottements dans les surfaces de contact le schiste se transforme en argile, matière dont la déformation en fonction du temps a été l'objet de maintes études.

L'effet du temps explique aussi que toute la pression n'est pas exercée sur le soutènement compressible au moment où il est placé, mais qu'à la longue les supports auraient à porter toute la pression statique  $\sigma = \gamma z$  du terrain situé en aplomb. Il y a donc un intérêt éminent de poursuivre l'exploitation du charbon sans interruption.

Pour éviter un malentendu il faut redire que l'application de la théorie de l'équilibre dans les massifs à frottement interne pour le calcul de l'inclinaison des surfaces d'affaissement était faite pour le massif qui s'étend infiniment dans les sens des  $X$ .

Si l'on exploite un chantier rond, l'affaissement dans le terrain incohérent se produit selon un cylindre en aplomb du vide créé.



Pour terminer ce paragraphe, qu'une observation de nature géologique soit permise.

Les matières rocheuses deviennent plastiques quand la pression dans toutes les directions est élevée. On peut admettre que dans l'écorce ter-

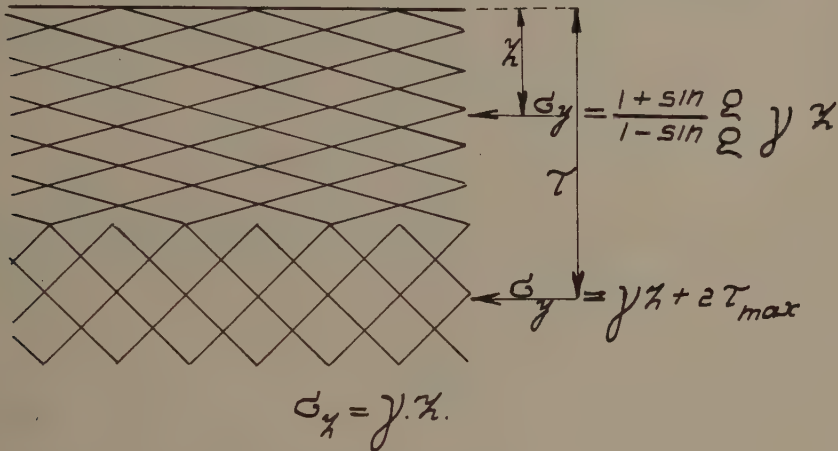


Fig. 12. Lignes de glissement et formules des pressions dans l'écorce terrestre comprimée.

tre, à un endroit où elle est en forte compression, la roche soit „plastique” à des profondeurs de plus de 10.000 mètres.

La figure 12 indique les stries de glissement ainsi que les formules pour les tensions horizontales pour la partie incohérente et pour la partie plastique,  $\gamma$  étant le poids spécifique,  $\varrho$  le talus naturel des débris de roche enchevêtrés et  $\tau_{max}$  la résistance au cisaillement de la roche plastique. Pour  $\varrho = 60^\circ$  (dans le gneiss cassé p.e.) les stries dans la masse incohérente s'entrecroisent sous un angle de  $30^\circ$ .

On calcule que la pression horizontale est différente dans les deux espèces de matière.

Dans la masse incohérente elle est beaucoup plus grande que dans la massa plastique. Il faut concevoir la formation des montagnes comme un effet de plissement, mais l'effort horizontal ne doit pas être calculé avec les formules de l'élasticité, mais selon les lois de l'équilibre pour les matières à frottement interne.

Les étirages dans l'écorce de la terre donnent peu de résistance dans la partie incohérente  $\left( \sigma_y = \frac{1 - \sin \varrho}{1 + \sin \varrho} \gamma z \right)$  et donnent lieu à la formation de plaines.

**Anatomy.** — *Neurobiotactic influences in the arrangement of midbrain and 'tween-brain centres.* By C. U. ARIËNS KAPPERS.

Dedicated to the memory of CLARENCE LUTHER HERRICK and to CHARLES JUDSON HERRICK on the fiftieth anniversary of the foundation of the Journal of Comparative Neurology.

(Communicated at the meeting of January 25, 1941.)

The influence of the direction from which stimuli reach ganglioncells, on the location of these cells, so evident in the motor nuclei of the bulb and in cell groups connected with the acoustic system, is also demonstrated in higher centres, especially in the mid- and tweenbrain of Teleosts, so well described already by C. L. HERRICK ('92).

A striking example is the development of the *torus longitudinalis*.

In lower fishes such as the Plagiostomes the roof of the midbrain shows a very simple structure, as a symmetric vaulting over the spacious midbrain ventricle or ventriculus opticus. In the dorsal midline, where both sides meet, this roof is thinner than laterally. Already in Ganoids, (Acipenser, JOHNSTON '01 and Polyodon, HOCKE HOOGENBOOM, '16) but especially in Teleosts a symmetric intraventricular longitudinal ridge of small cells appears under the midline, which acquires its greatest volume over the posterior commissure (Megalops and Monopterus, fig. 1), decreasing gradually in a backward direction and finishing some distance before the caudal end of the optic ventricle (Periophthalmus, fig. 2).

This structure, derived from the deeper parvocellular elements of both halves of the tectum, is not equally thick in all Teleosts. The midbrain roof being the most important centre of optic impulses, FRANZ ('12) was inclined to believe that the degree of development of the torus longitudinalis depends on the size of the optic nerve or optic tectum. He based this opinion on the small size of this torus in a blind fish (Trypauchen) and its large size in Raniceps.

Direct connections of the torus longitudinalis with the optic nerve, however, have never been observed, and — although this torus may be quite large in some fishes with large eyes (c.f. Megalops, fig. 1), in several fishes with even larger optic nerves, such as Orthogoriscus mola (c.f. also BURR, '28), Exocoetes evolans, and Periophthalmus, the torus is even less developed than in Monopterus and Raniceps. Besides, according to our ('06), WALLENBERG's ('13) and JANSEN's ('29) experience, the size of the torus longitudinalis is not in the least influenced by atrophy of an eye and amongst the blind fishes examined by CHARLTON the torus is quite large in Troglichtys rosae ('33, fig. 8). Consequently a direct relation between the size of the torus and visual projection does not exist.

Which then is the factor that determines its development? First of all



I wish to emphasize that this subtectal mass develops from the medial part of the deep parvocellular layer of the tectum (SARGENT, '03), whereas the optic fibres chiefly end in the superficial layers. Secondly I can confirm KUDO's statement ('23) that the torus longitudinalis receives a bundle of fibres from the cerebellum (tr. cerebello-tectalis or: tr. cerebello-toralis). After its origin from the corpus (= vermis) cerebelli, probably from PURKINJE cells, this bundle runs forward medially and dorsally to the lateral lemniscus joining the tecto-cerebellar tract (fig. 3, *Belone*) which originates from the fronto-lateral part of the tectum (see fig. 1, *Monopterus*) ending in the corpus cerebelli (cf. WALLENBERG '13). The toral fibres continue their course underneath the ventricle (see *Periophthalmus*, fig. 3) to enter the torus frontally and laterally to the commissura posterior (*Monopterus* fig. 1, similarly in *Raniceps*).

TUGE ('34b), who also traced a cerebello-tectal bundle (in addition to a larger tecto-cerebellar tract) in *Carassius auratus*, found the cerebello-tectal fibres in this animal mingling with the lemniscus bundle, to enter the lateral part of the tectum. "None of them were found farther orad." We could, however, trace some fibres in the rather small torus longitudinalis of this animal on the level of the commissura posterior, where both halves of the torus join. In fig. 5 of his '35 paper TUGE also draws these fibres. Caudally both halves of the torus are pushed wide apart by the pressure of the large valvula cerebelli on the tectum.

As another bundle that may influence the development of the torus longitudinalis we may consider a strand of fibres arising from the frontal subtectal region on the level of the commissura posterior. KUDO ('24), who was the first to trace this bundle, found it strongly developed in *Raniceps*. According to MEADER ('34), this bundle is not constant, but KUDO found it also in *Motella*.

The cerebello-toral fibres are strongly developed in *Monopterus* with its large torus and small tectum and also in *Raniceps* and *Megalops*. Most of them end in the homolateral, about one fifth or less in the contralateral torus half (see fig. 1: *Monopterus*).

The fact that this tract enters the torus in the region above the commissura posterior explains the greater size of the torus of this level. The rest of its fibres runs backwards in the torus, terminating gradually among its cells. These cells, first described by SALA ('95), are small and also because of the form of their short dendrites closely resemble the granular cells of the cerebellum. As already stated by KUDO ('23) in most fishes their axiscylindres are unmyelinated (see *Megalops*, fig. 1) or surrounded by some myelinated fibres only (*Scardinius*<sup>1)</sup>).

<sup>1)</sup> The fact that, wherever myelinated fibres run together with unmyelinated ones, they tend to acquire a sheathlike arrangement round the unmyelinated fibres is very striking also in the comm. habenularum of *Plagiostomes* and in the fasc. retroflexus of several animals (cf. KAPPERS, HUBER and CROSBY '36). They form a sort of sheath round the unmyelinated fibres (l.c. fig. 437) and it is not unlikely that this arrangement is to be explained by the same influence that acts in the peripheral accumulation of the myeline sheath.

In *Gobius* (fig. 1) only we found them provided with some myeline. As already stated by SALA ('95), RAMON ('99) and KUDO ('24) these axiscylindres run dorsally into the superficial layer of the tectum associating their impulses with those of the optic radiation and contributing considerably to the thickness of its superficial layer, especially in the medial part of the tectum (see fig. 1, *Gobius*). This is very evident also in *Raniceps* (KUDO). — Laterally the thickness of this superficial layer of the tectum decreases.

The prevailing transverse spread of these fibres also shows a certain resemblance to the transverse spread of the axiscylindres of the granular cells in the molecular layer of the cerebellum; with this difference, however, that the torus fibres do not dichotomize. Even the topographic relation of the torus longitudinalis to the tectum, strongly resembles the topographic relation of the granular masses to the molecular layer as found in the cerebellum of *Plagiostomes*<sup>1)</sup>.

Although the spatial economy of the Teleostean brain case in larval life may favour the intraventricular outgrowths in Teleosts<sup>2)</sup>, it is evident that the direction, location and degree of this outgrowth is determined by the influence of the fibres reaching it from the ventricular side, which also explains the greatest development of the torus at the level where these fibres enter it.

Whereas the intraventricular outgrowth of the torus shows the neurobiotactic influence of tecto-petal fibres arising in the cerebellum, the development of the *valvula cerebelli* shows the same influence exercised by cerebello-petal fibres originating from the midbrain. Contrary to *Plagiostomes* in most Teleosts (and in Ganoids, JOHNSTON '01, HOCKE HOOGENBOOM, '16) a part of the cerebellum grows out underneath the midbrain roof pushing forward the thinned hindwall of the tectum.

This *valvula cerebelli* derives from the basal part of the corpus (= vermis) cerebelli. The size of the subtectal part or *valvula* varies greatly in different Teleosts. In some fishes such as *Lophius*, *Periophthalmus* (fig. 2) and *Hippocampus*, it only consists of one impair lobule. In others, the number of impair lobuli increases (e.g. *Perca*, fig. 2), and in others again paired lobuli develop on each side of the impair one filling up the posterior part of the optic ventricle. (*Belone*, fig. 2.)

<sup>1)</sup> This cytological resemblance between distant territories with related functions is not exceptional. The typical structure of the "cerebellar crest" covering the bulbar centres of the lateral line organs and the resemblance of the protopathic substantia gelatinosa in the spinal cord, the spinal trigeminus nucleus and the substantia gelatinosa accompanying the solitary tract in man are other examples of homologous functions determining homologous structures in different regions. A similar observation is made by DE CRINIS for mammals (Journ. f. Psych. und Neur., 1934, 45).

<sup>2)</sup> Spatial economy acts a part also in the development of the forebrain in these animals as it does in birds.

The frontal direction and the degree of this outgrowth is determined by a fibre tract reaching the cerebellum chiefly from the nucl. lateralis valvulae, the tr. tegmento-cerebellaris (tr. teg. cer. fig. 2). This nucl. lateralis valvulae (n. lat. val.) is intimately related to end stations of the lateral lemniscus; the torus semi-circularis and ganglion isthmi, and to HERRICK's anterior gustatory nucleus. Contrary, however, to higher vertebrates the lateral lemniscus of fishes has to do chiefly with stimuli resulting from the lateral line organs, which are not concerned in hearing but which serve a static function. For this static function the tegmentum mesencephali is the chief projection and correlation centre. From it impulses are conducted chiefly to the basis of the cerebellum, which also receives some direct lateral nerve fibres (ADDISON, '23; PEARSON, '36).

An other bundle, not reaching the valvula but the corpus cerebelli from a frontal centre is the tecto-cerebellar tract, whose cerebellopetal course has been proved by WALLENBERG ('13) and a smaller bundle arising from the ganglion isthmi — a static centre intercalated between the tectum and corpus cerebellum. Of all these systems the tegmento-mesencephalic tract, however, sends its fibres into the valvula cerebelli and the intraventricular outgrowth of the valvula, specially the development of its lateral lobes runs parallel to the development of this system <sup>1)</sup> (Belone, fig. 2). This is most conspicuous in such fishes as the Cyprinoids and Siluroids (HERRICK '05) whose gustatory system is so strongly developed and in Mormyridae, whose lateral nerves are hypertrophic and whose valvula cerebelli acquires an enormous size (FRANZ '11, STENDELL '14, BERKELBACH VAN DER SPRENKEL '15, SUZUKI 32c).

In addition to these two instances of neurobiotactic influence on the configuration of the midbrain I shall mention two examples of this influence in the diencephalon. The first of these concerns the development of the *nucleus rotundus*, a prominent nucleus in the ventral thalamus of most Teleosts, first described by FRITSCH ('78) and whose relations to an other thalamic nucleus, the *nucleus anterior*, has been elucidated by FRANZ ('12), who was the first to show that these two nuclei are closely related structures and who — on account of their internal glomerular structure, first noted by BELLONCI ('85) and CATOIS (l.c. '02, p. 104, fig. 27) — classified them together under the name of corpus glomerulosum. Both nuclei are characterized by a number of central glomeruli consisting of axonic endfeet of various origin, synapting with tufts of dendrites arising from peripheral cells.

The nucleus anterior of Teleosts, which has nothing in common with the homonymous nucleus of mammals, is located dorso-frontally under the

<sup>1)</sup> This system connecting the valvula cerebelli with the nucl. lateralis valvulae should be well distinguished from the brachium conjunctivum or tr. cerebello-tegmentalis which arises farther backward in the corpus cerebelli, ending (after having crossed) in the basis of the mid- and 'tweenbrain.



frontal part of the tectum, medially to the posterior pole of the lateral geniculate nucleus, with which it is connected by internuclear fibres (HOLMGREN, '20; MEADER, '34) and laterally to and behind the region of the medial optic ganglion described by WALLENBERG ('13) and also observed by MEADER ('34, p. 378). The nucleus anterior itself does not receive optic fibres<sup>1</sup>). Dorsally to the nucleus anterior most fibres of the commissura horizontalis arise and end. This commissure is also connected with the caudoventral or rotundus part of the corpus glomerulosum, which practically is intercalated in this bilateral connection between the frontal tectal (and pretectal) areae of the midbrain, the projection centres of the frontal visual field (LUBSEN, '21; STRÖER, '39), so important for the capture of food.

According to FRANZ' researches the nucleus anterior is the phylogenetically oldest part of the corpus glomerulosum. In Physostomes, where the nucleus rotundus part of the corpus glomerulosum fails, it is very large (see also JEENER, '30). Among the Physoclists the rotundus part may fail or be little developed in the Anacanthini. In Acanthopterygi the rotundus part acquires its greatest extension, while the dorso-frontal anterior nucleus decreases.

We have been able to reexamine the relations of the corpus glomerulosum system in a great number of Teleosts. Our results largely agree with FRANZ'.

In *Amia*, *Lepidosteus*, and many Physostomes (pike, herring, carp, eel, *Silurus*) the anterior nucleus is well developed, while a glomerular rotundus nucleus as it appears in the higher Teleosts in the caudo-ventral part of the thalamus, is absent. A glomerular rotundus nucleus does not, however, fail in all Physostomes<sup>2</sup>). In *Megalops* it is even very large.

Among the Anacanthini which we examined it is present in *Exocoetes*, *Scomber*, *Ctenolabrus*, *Gadidae* and *Pleuronectidae*. In the *Acanthopterygii* the rotundus part is well developed, in the *Plectognath* *Tetrodon* and in the *Lophobranchs* *Syngnatus* (see fig. 3) and *Hippocampus* it attains its greatest size.

The nucleus anterior in these animals is much reduced and what is left of it is connected by a stalk-like elongation with the glomerular substance of the nucleus rotundus (see fig. 3, *Syngnatus*), as described by FRANZ.

While the decrease of the anterior nucleus accompanying the increase of the rotundus nucleus suggests that the development of the rotundus takes place at the expense of the anterior, the question arises what is the

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<sup>1</sup>) According to FRANZ ('12), HOLMGREN ('20) this nucleus is also connected with the commissura minor of *C. L. HERRICK*, which may act a similar part in connection with the anterior nucleus as the comm. horizontalis does in connection with the rotundus nucleus.

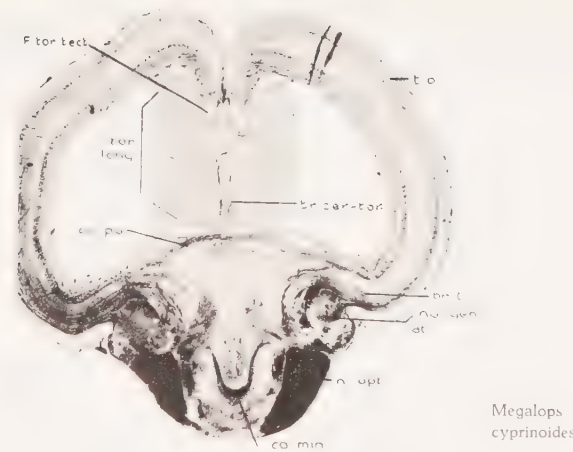
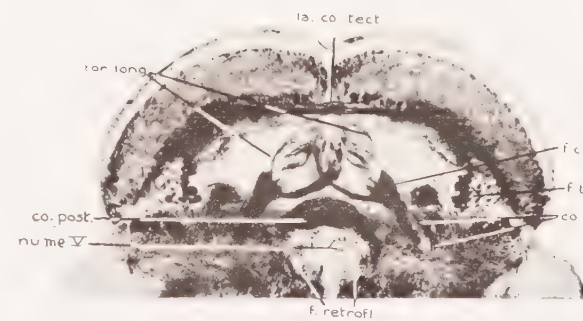
<sup>2</sup>) It may not be superfluous to state that the air-bladder itself has nothing to do with this nucleus which is very large even in the *Pleuronectidae* where the air-bladder is atrophic. *Symbranchus* whose airbladder is equally atrophic also possesses a rotundus nucleus.

## C. U. ARIENS KAPPERS: NEUROBIOTACTIC INFLUENCES IN THE ARRANGEMENT OF MIDBRAIN AND 'TWEEN-BRAIN CENTRES.

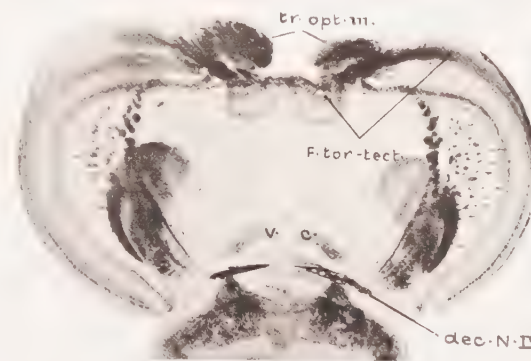
of CLARENCE LUTHER HERRICK and to CHARLES JUDSON  
the fiftieth anniversary of the Journal of Comparative Neurology

sections of the tectum opticum and torus longitudinalis of *Megalops* *Monopterus javanensis*. Horizontal section of the tectum and torus longitudinalis of *Gobius speciosus*.

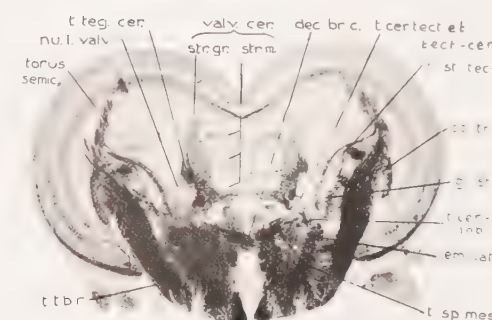
tecti (- fibr. geniculo-tectales); co. h. - comm. horizontalis;  
 minor (C. L. HERRICK); co. po. - comm. posterior; dec. N. IV  
 cerebello-toralis; f. retrofl. - fasc. retroflexus;  
 tectales; f. t. c. - fasc. tecto-cerebellaris; la. co. tecti = lamina  
 opticus; nu. gen. lat. - nucleus geniculatus  
 V = nucleus mesencephalicus trigemini; t. o. - tectum opticum; tor  
 gitudinalis; tr. - tractus cerebello-toralis (- f. c. t. - fasc. cerebello

Megalops  
cyprinoides

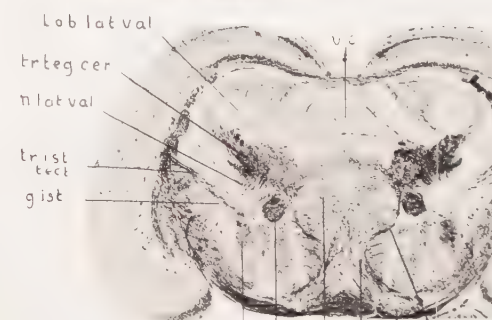
Monopterus  
javanensis



Golubus



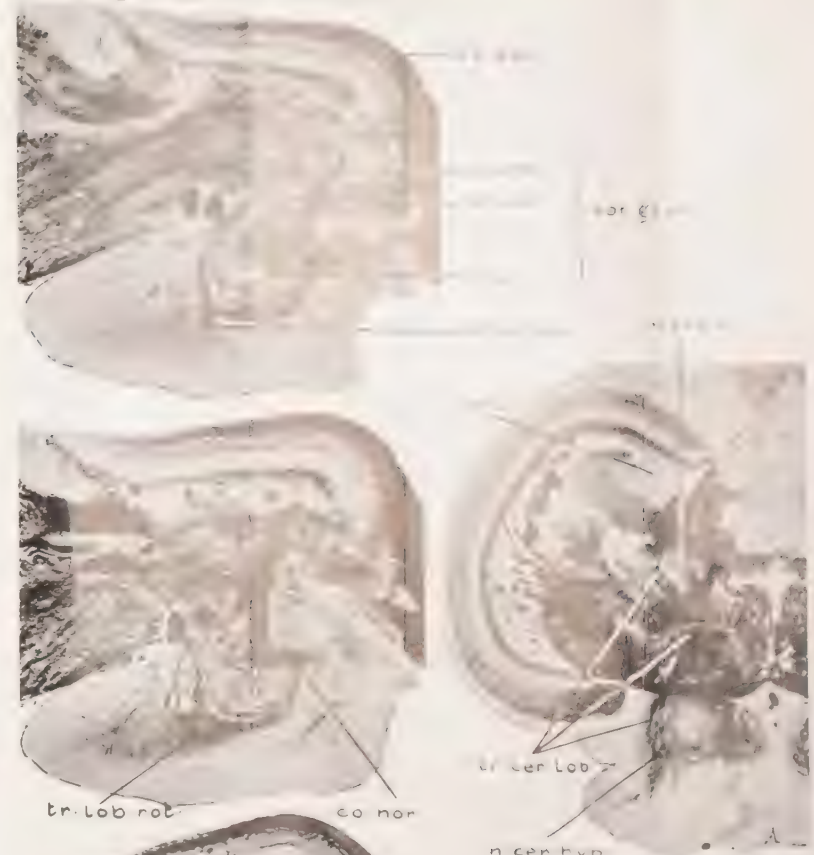
Perca  
fluviatilis



Belone  
belone



Syngnathus acus.



Scardinius  
erythrophthalmus.

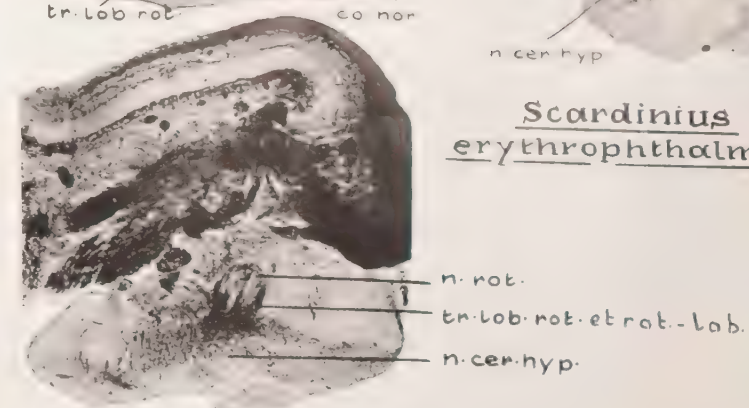


Fig. 3





reason of the caudo-ventral displacement and accumulation of this glomerular system?

In connection with this we must realize that the ventro-caudal accumulation of glomeruli resulting in the formation of the rotundus nucleus takes place in the caudal end of a strand of cells formerly described by us as *nucleus prerotundus* because this column commences at some distance in front of the rotundus region. It, however, is not only in this sense that the name prerotundus is appropriate to this strand of cells, the name happens to be appropriate also in so far as its caudal pole serves as a basis for the formation of the rotundus nucleus. The small cells of the nucl. prerotundus (also in its caudal pole) receive fibres from the tr. olfacto-hypothalamicus lateralis.

The first form in which the glomerular rotundus nucleus develops is such that a small number of glomeruli appear amongst the small cells in the caudal pole of the prerotundus nucleus. This arrangement is seen in some Physostomes (carp, c.f. also SHELDON, '12) and also in the Physoclist, *Ammodytes tobianus*.

In the higher fishes the number of these central glomeruli in the rotundus nucleus increases, and the cells move more and more to the periphery, their dendrites entering the glomerular centre where they synapt with the afferent olfactory fibres, with tecto-rotundus fibres and with fibres of the horizontal commissure, originating on the contralateral side. In several Acanthopterygei especially in Plectognathi and Lophobranchi the small cells have nearly all migrated to the periphery forming a cellular capsule around the glomerular centre. This gradual accumulation of the small cells, part of which may have shifted from the anterior nucleus to the periphery of the glomerular mass of the rotundus has been observed also in Japanese fishes by our former collaborator, Prof. SUZUKI ('32 a).

While the dendrites of these cells enter the glomerular centre, their axiscylindres run downward and backward ending in the hindpole of the lobi inferiores <sup>1)</sup>. Similar exones arise from the peripheral cells of the nucl. anterior, forming a thalamo-lobar tract. Contrary, however, to the nucleus anterior the nucl. rotundus also receives a large number of fibres arising in the lobi inferiores and ending among the central glomeruli (see also HOLMGREN '20). These fibres <sup>2)</sup> originate in a large mass of cells located in the postero-lateral part of the lobi inferiores, rightly described by GOLDSTEIN ('05) as nucl. cerebellaris hypothalami (fig. 3 n. cer. hyp.). The chief afferent connection of this nucleus is obviously made by a tract that

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<sup>1)</sup> SUZUKI ('32 a) rightly observed that this tr. rotundo-lobaris is best developed in such fishes where the peripheral cell mass of the rotundus is well developed.

<sup>2)</sup> HOLMGREN ('20) considers these fibres as homologous to the mammillo-thalamic tract (Vicq d'Azyr) of Mammals, an opinion which we cannot share since the caudo-lateral part of the lobi inferiores, where most of these fibres arise, is by no means to be compared with the corpus mammillare. It does not receive fornix fibres but fibres from the nucl. lateralis valvulae in front of HERRICK's secondary gustatory nucleus.

arises from the cerebellum and the nucleus lateralis valvulae, immediately in front of HERRICK's secondary gustatory nucleus (the Rindenknotten of German authors). In our microphotograph of *Scardinius* (fig. 3) the whole course of this tract, already observed by EDINGER ('96) and HERRICK ('05, p. 420) is seen in one section and is labelled as tr. cerebello-lobaris (tr. cer. lob.). This connection, which may run both ways<sup>1</sup>), is very distinct also in HOLMGREN's figs. 87, 88, 89 of *Callionymus* and in SUZUKI's picture of *Plécoglossus altivelis* (32 b., fig. 6).

It includes also fibres of the tertiary gustatory tract described by C. J. HERRICK ('05) but the fact that the nucleus lateralis valvulae is so closely connected with the cerebellum and also sends descending fibres in the fasc. long. posterior (tr. n. valv. desc. in Belone, fig 2) makes it evident that the cerebello-hypothalamic tract has a prevailing static function.

We do not believe that this static function has to do with gustatory impulses only. We are more inclined to assume that the location of the small celled secondary gustatory nucleus immediately behind the nucl. lateralis valvulae is an expression of the fact that in fishes, where taste buds are spread over a large part of the body, taste also has a directive influence. HERRICK himself ('05) emphasized the close relation of his secondary gustatory nucleus to the nucleus lateralis valvulae (l.c. p. 419 and 452) and of his tertiary gustatory tract to cerebello-lobar and lobo-cerebellar fibres. The addition of gustatory impulses to this tract may explain that this tract has its greatest development in Cyprinoids and Siluroids where taste is most developed. (ADDISON, '23) and where the nucl. lateralis valvulae and the valvula cerebelli are also very large.

I therefore believe that the addition of static projections to the lobi inferiores has to be considered chiefly responsible for the development of the lobo-rotundus system and the caudo-ventral displacement of the glomerular body, the more so as the lobi inferiores also receive a descending geniculate bundle. This may also explain that the lobo-rotundus tract and the nucleus rotundus are well developed in Megalops, and Lophobranchi whose gustatory system is not nearly so strongly developed as in Cyprinoids and Siluroids.

So much is sure that — whereas the fixation of the dorso-frontal part of the corpus glomerulosum (or nucleus anterior) may be explained by its connections with the pretectal and anterior tectal area — the increase of the glomerular system in the caudo-ventral rotundus nucleus partly at the expense of nucl. anterior has to be explained by the increasing influence of the caudo-lateral part of the lobi inferiores on this system, and the structural accommodation to these impulses in the more differentiated Teleosts.

The various stimulations correlated in the rotundus part of the corpus glomerulosum are affected, partly by lobo-cerebellar fibres, partly by the lobo-bulbar fibres arising in the hypothalamus and ending on the level of the trigeminus nucleus (basi-quintal tract of WALLENBERG, '31). According to HOLMGREN ('20) collaterals of the tr. lobo-rotundus also take a descending course.

<sup>1</sup>) Cf. WALLENBERG '07, p. 37 and WALLENBERG '31, p. 260—261.

The last example of a shifting to be mentioned here concerns the *hypothalamic hypophysary nuclei*. As is well known in Mammals the hypophysary nuclei may be divided into three groups, a basal supra-optic and paraventricular magnocellular nucleus and some parvocellular tuberal groups, the former being located in the preinfundibular, the latter in the postinfundibular region of the hypothalamus. Of the two magnocellular nuclei the supraoptic one is chiefly concerned with the hypophysary function, influencing water- and salt metabolism.

Ontogenetically and phylogenetically both magnocellular nuclei derive from a continuous mass of large cells, the so-called preoptic magnocellular nucleus of fishes. From the descriptions by JOHNSTON ('01), RÖTHIG ('11), SHELDON ('12), CHARLTON ('32), KAPPERS ('34) and BOON ('38) it appears that in its primitive form this nucleus presents itself as a mass of magnocellular elements, located in front and above the optic chiasm.

In some Teleosts a part of the cells of this nucleus shifts backward along the hypothalamo-hypophyseal bundle to acquire a far more caudal position near the hypophyseal stalk (Gadus, RÖTHIG, '11; Sarda, CHARLTON, '32; Belone, KAPPERS, '34). In Mammals (rabbit, dog, apes and man) also a part of the basal hypophyseal nucleus shifts over the optic chiasm to form a postchiasmatic parahypophyseal nucleus.

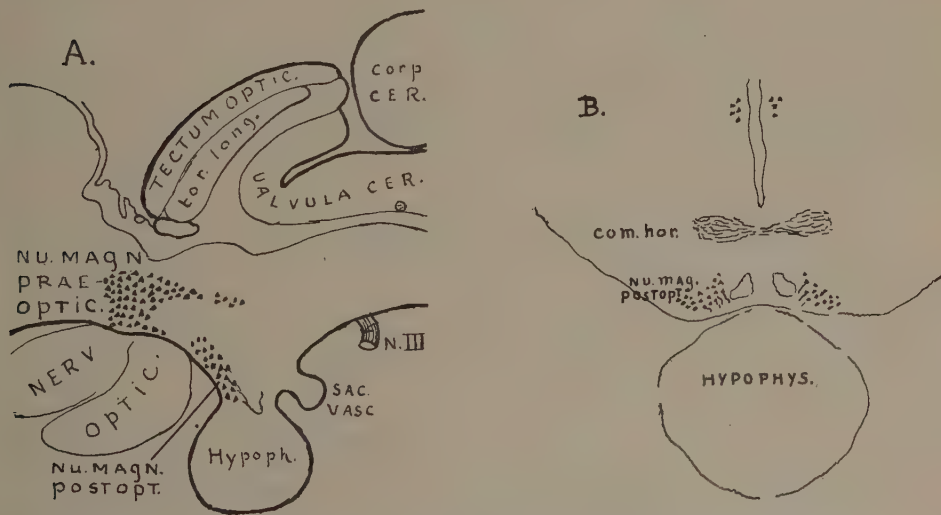


Fig. 4. A. Sagittal section of the brain of *Sarda*, after CHARLTON, showing the location of the preoptic and postoptic magnocellular nuclei. B. Transverse section of the brain of *Belone* showing the location of the postoptic magnocellular nucleus on the level of the hypophysis.

The shifting of these cells in the direction of their effector, the hypophysis, is a most interesting phenomenon. Whereas other migrations in the central nervous system, proceed in the same direction in which the dendrites of their cells (the chief receptors of the impulses) grow out, the



shifting of this hypophysary nucleus apparently occurs in the direction of its axiscylindres, thus approaching the effector, the hypophysary gland.

A similar migration in an axonic direction is observed in the onto- and phylogenetic development of the autonomic system of the viscera. It is a well known fact that the postganglionic cells of the sympathetic chain develop either from the neural crest (MÜLLER and INGVAR, '23), or, more probably from the spinal cord itself along the ventral roots (KUNTZ and BATESON '20, JONES, '37) or both ways (KUNTZ, '29; TERNI, '31). Whatever may be the case, all postganglionic cells in the embryo (as in adult primitive vertebrates) are located near the spinal cord or bulb. Later in development (similarly in higher vertebrates compared with lower ones) an ever increasing number of the cells shifts into the direction of their effectors and, migrating along the axonic fibre strands, acquires a juxta-mural or an intramural position.

Since this proces of migration apparently is the opposite of what usually occurs in the central nervous system, there must be a special reason for it. This reason is to be found in the different ways in which reflexes are effected in the central and autonomic nervous systems. Whereas in the central nervous system reflex action occurs through the transmission of the stimulus to the dendrites (or body) of the cells, LANGLEY and ANDERSEN ('94) pointed out that the majority of the reflexes in the visceral autonomic system are *axon reflexes*, i.e. reflexes initiated by impulses originating in the effector. Reaching the postganglionic cells by way of their axons these impulses spread again to the effector by means of collaterals. These anti-dromic or axon reflexes according to our opinion ('08) are responsible for the antidromic shifting of autonomic cells towards their effector.

In his lecture on the autonomic nerves given at *Amsterdam* ('05) LANGLEY said: "from the axon reflex we naturally pass to consider whether any reflex in the usual sense occurs in the other visceral ganglia". He then further stated: "No reflex action is obtained from them which cannot reasonably be referred to an axon reflex" (l.c. p. 16). — Although this may be exaggerated, the prevalence of axon reflexes in the sympathetic nervous system is very likely also owing to the fact that whereas in the somatic roots the number of sensory fibres usually exceeds the number of motor fibres, the number of sensory visceral fibres in the rami albi is far less than of the preganglionic ones, (1:8 approximately). — Considering the fact that the number of postganglionic fibres again exceeds at least thirty times the number of preganglionic ones (RANSON and BILLINGSLEY, '18) it is not at all surprising that most reflexes occurring in the viscera are axonic ones, thus explaining also the migration of the postganglionic cells in the direction of the effector.

Also STIEMENS ('34) in her work on the phylogenetic development of the vagus and sympathetic system considers the antidromic reflex as responsible for the increasing peripheral displacement of thoracic and abdominal postganglionic cells in the series of Vertebrates, in the direction of their effectors, a process that runs parallel with the phylogenetic increase of intestinal activity.

Since, however, the hypothalamic cells referred to are also to be con-

sidered as autonomic cells, comparable to postganglionic visceral elements, their migration in the direction of the hypophysis confirms our explanation ('08) of the peripheral migration of the postganglionic cells of the viscera.

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**Chemistry.** — *On the Resolution of  $\alpha$ -Phenylpropylamine into Its Optically-active Antipodes* (2nd Part). By F. M. JAEGER and W. FROENTJES.

(Communicated at the meeting of January 25, 1941.)

§ 1. In a former paper<sup>1)</sup> the preparation of this base and that of its someride *benzedrine*, as well as the resolution of the latter into its antipodes, have been described in detail. In the present one we communicate the results of our investigations concerning the resolution of  *$\alpha$ -phenylpropylamine*:  $C_6H_5 \cdot CH(NH_2) \cdot CH_2 \cdot CH_3$  and of the phenomena therewith observed.

Purest racemic  *$\alpha$ -phenylpropylamine* of the boilingpoint  $62^\circ$ — $63^\circ$  C. under 3 m.M. pressure was converted into its *bi-d-tartrate* and the anhydrous salt obtained subjected to repeated fractional crystallisations from *absolute* alcohol, — first at room-temperature and later in an exsiccator at  $35^\circ$  C., — this being the most adequate temperature for still obtaining well-crystallized fractions from the last mother-liquors. The principal fraction (of  $[\alpha]_D = +22^\circ$ , 5) was thus obtained from the solution at  $18^\circ$  C. and recrystallized from absolute alcohol; but it proved impossible also to isolate the final fraction (of  $[\alpha]_D = +7^\circ$ ) from the mother-liquors in this way. Therefore, — as no other solvent proved to yield better results, we finally made the experiments at  $35^\circ$  C. in the way described. As the difference in solubility of the components proves to be much less than in the case of *benzedrine*; and because their power of crystallisation is rather poor, — the complete resolution happens to be a much more tedious task and involves a greater sacrifice of material than in the case of *benzedrine*; the more so, because the *bi-tartrates*, which in *cold* alcohol are only sparsely soluble, could never be obtained in the form of well-developed crystals, notwithstanding many tentatives were made and after other solvents had been tried. They always were deposited as felt-like, very thin and feathery needles or, from the final mother-liquors, as a slimy aggregation. Moreover, the substance has a disposition to “creep” against the walls of the vessel to a considerable distance above the surface of the solution.

Finally a fraction could be obtained which, after repeated crystallisations, did not change its specific rotation for sodium-light any more. This constantly rotating fraction showed  $[\alpha]_D = +22^\circ,5$ ; its meltingpoint was  $176^\circ$  C.<sup>2)</sup> The base set free from this salt (boiling at  $67^\circ$  C. under

<sup>1)</sup> F. M. JAEGER and J. A. VAN DIJK, Proc. Ned. Akad. v. Wetensch., Amsterdam, **44**, 26 (1941).

<sup>2)</sup> P. BILLON, Compt. rend. Paris, **182**, 472 (1926). The meltingpoint:  $176^\circ$  C. is  $10^\circ$  higher than that given by BILLON ( $166^\circ$  C.); this again demonstrates the incompleteness of the fission by this author.



5 m.M. pressure), proved to be the *levogyrotory* component. The rotations observed were *less than half* those of *benzedrine*; but they were *not less than 50 % higher* than those indicated by BILLON<sup>2)</sup>, whose resolution evidently must have been still very *incomplete*. The mother-liquors of the final recrystallisations of the mixed *bi-d-tartrates* did not crystallize very well and yielded a kind of semi-colloidal product; but a final fraction thus obtained, showing a (lowest) specific rotation:  $[\alpha]_D = +7^{\circ}.0$  proved to be *homogeneous* and, on further recrystallisations, did no longer alter its rotation any more. This *bi-d-tartrate* showed a meltingpoint of  $123^{\circ}$  C.; and the base set free from it, proved, indeed, to be the *dextrogyrotory* component, constantly, boiling at  $64^{\circ}$  C. under a pressure of 4 m.M. The rotatory dispersions of the two antipodes thus observed are given in Tabel II, whilst the same has been done for the *bi-d-tartrates* themselves in Table I.

TABLE I.

The Rotatory Dispersion of *l-a*-Phenylpropylamine-*bi-d*-Tartrate ( $176^{\circ}$  C.) and of *d-a*-Phenylpropylamine-*bi-d*-Tartrate ( $123^{\circ}$  C.) in 2% aqueous Solutions.

Wave-length $\lambda$ in A.U.:	Specific Rotations $[\alpha]$ of	
	<i>l-a</i> -Phenylpropylamine- <i>bi-d</i> -tartrate ( $176^{\circ}$ C.):	<i>d-a</i> -Propylamine- <i>bi-d</i> -tartrate ( $123^{\circ}$ C.):
6980	$+16^{\circ}.0$	—
6730	17.8	$+5^{\circ}.2$
6480	18.8	5.9
6262	19.8	6.1
6074	20.8	6.5
5893	22.3	7.0
5735	23.6	7.2
5592	24.5	7.7
5463	25.8	8.0
5340	27.1	8.0
5224	28.1	8.0
5126	29.3	8.1
5036	30.1	—
4950	31.5	8.3
4861	32.4	—
4793	35.0	—
4724	36.8	—
4596	39.0	—
4537	42.1	—
4483	(47.0)	—

<div>TABLE II.</div> <div>The Rotatory Dispersion of Optically-active <math>\alpha</math>-Phenyl-propylamine.</div>			
Wave length $\lambda$ in A.U.:	Specific Rotations of		Mean values of the Dispersion:
	<i>d</i> - $\alpha$ -Phenyl- propylamine	<i>l</i> - $\alpha$ -Phenyl- propylamine	
6980	—	—11.7	(11.7)
6730	+11.4	—12.2	11.8
6480	+12.2	—12.7	12.5
6262	+13.0	—13.3	13.2
6074	+13.9	—14.2	14.1
5893	+14.8	—15.1	15.0
5735	+15.7	—16.0	15.9
5592	+16.7	—16.9	16.8
5463	+17.6	—17.9	17.8
5340	+18.4	—18.7	18.6
5224	+19.2	—19.5	19.4
5126	+20.2	—20.4	20.3
5036	+20.9	—21.2	21.1
4950	+21.9	—23.0	22.5
4861	—	—23.8	23.8
4793	—	—24.7	24.7
4724	—	—25.5	25.5
4658	—	—26.3	26.3
4596	—	—27.2	27.2
4537	—	—28.0	28.0
4483	—	—28.8	28.8

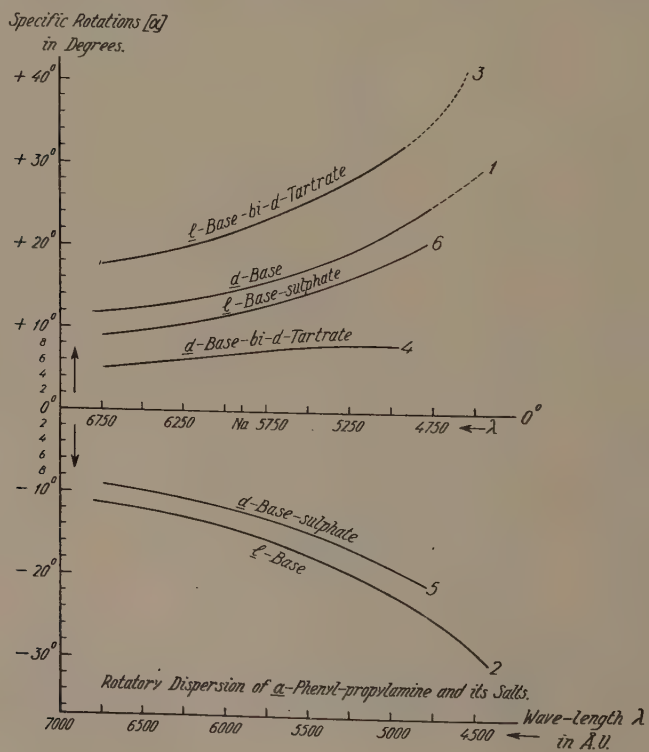


Fig. 1. Rotatory Dispersion of  $\alpha$ -Phenylpropylamine and its Salts.

§ 2. From these data it becomes clear that the two optically active bases, when present in the form of the *bi-tartrates* exhibit a *rotation of the opposite algebraic sign* as compared with that of the free bases themselves: they thus manifest this phenomenon, when they are electrically charged as *ions*. For the purpose of corroborating this conclusions, we have prepared the (anhydrous) *sulphates*<sup>1)</sup> and in Table III we give the corresponding data of the rotatory dispersion for these salts in a 2 % molar (= 1,842 % in weight) aqueous solutions:

TABLE III. The Rotatory Dispersion of the Sulphates of the Optically-active $\alpha$ -Phenylpropylamines in aqueous Solutions,		
Wave-length $\lambda$ in A.U.:	Specific Rotations $[\alpha]$ of $\alpha$ -Phenylpropylamine-sulphates:	
	<i>l</i> - $\alpha$ -Phenylpropylamine-sulphate	<i>d</i> - $\alpha$ -Phenylpropylamine-sulphate
6730	+ 9.4	-10.0
6480	+10.5	-10.7
6262	+11.4	-11.5
6074	+12.0	-12.0
5893	+13.2	-13.5
5735	+14.0	-14.0
5592	+14.4	-14.7
5463	+15.3	-15.3
5340	+16.2	-16.2
5224	+17.2	-16.7
5126	+18.1	-17.2
5036	+19.2	-17.7
4950	+20.2	-18.3
4861	+20.8	-19.5
4793	+21.7	-20.5

<sup>1)</sup> Analysis yielded 25.82 %  $\text{SO}_4$  (calc.: 26.07 %); the salt melts at 200° to 223° C. under partial decomposition. The results, which are graphically represented all-together in Fig. 1, leave no doubt whatsoever about the correctness of the conclusion drawn. The behaviour of the bases, therefore, is in this respect exactly the *opposite* one to that of the isomeric *benzedrine*. Probably the sterical configuration round the asymmetrical carbon-atom in the two isomeric substances may be supposed to be enantiomorphous.



**Chemistry.** — *The Temperature- Coefficient of the Electrical Resistance of Ruthenium and its Thermo-electrical Behaviour with respect to Platinum.* By F. M. JAEGER and E. ROSENBOHM.

(Communicated at the meeting of January 25, 1941.)

§ 1. From our measurements of the specific heats of *ruthenium* we, in a paper published in 1932, could demonstrate that this metal most probably occurs in at least *four* different solid states, distinguished by us as  $\alpha$ -,  $\beta$ -,  $\gamma$ - and  $\delta$ -*ruthenium*, which are consecutively transformed into each other in a perfectly reversible way at the transition-temperatures: 1055°, 1200° and 1500° C. respectively. Although this element, — being paramagnetic, — has no CURIE-point, the behaviour of the metal appears in many respects to be analogous to that of its preceding group-homologue: *iron* <sup>1)</sup>).

It seemed desirable to corroborate the said deductions by other experiments; but the chief difficulty arose from the fact, that *ruthenium* is an almost mechanically *unworkable* material, because of its extreme hardness, its brittleness, its oxydability and its high meltingpoint (1966° C.). Ordinarily it is obtained as a grey powder; and neither can it be drawn into wire-form, nor be shaped into a homogeneous bar or hammered into plates.

By the kind help of the firm of Dr. HERAEUS in Hanau, we had at our disposal a small bar of about 60 m.M. length and with a rectangular cross-section of about 13 m.M<sup>2</sup>., which was prepared by strong compression of the purest, powdery metal and by subsequent sintering in a high vacuum at a very high temperature. Although the density of this sample proved to be only about 92 % of the true value for the molten and solidified metal, the bar appeared finely crystallized, not too inhomogeneous and sufficiently coherent for allowing a determination of the relative temperature-coefficient of the electrical resistance, heat-capacity, etc. Its specific resistance proved to be:  $7 \cdot 10^{-6}$ , — this at least being of the same order of magnitude as the value given in the literature <sup>2)</sup>:  $7,6 \cdot 10^{-6}$ . Measurements of the *absolute* resistances at different temperatures were, of course, beforehand excluded; but as we chiefly were interested in the dependence of the resistance on the temperature, this fact seemed in this case only of secondary importance.

§ 2. The measurements of the electrical resistance, which presented considerable difficulties, were made in the way already previously described <sup>3)</sup>, i.e. by means of the method of the optically-coupled double-

<sup>1)</sup> F. M. JAEGER and E. ROSENBOHM, Rec. d. Trav. d. chim. d. Pays-Bas, **51**, 32, 45 (1932).

<sup>2)</sup> A. E. VAN ARKEL, *Reine Metalle*, Berlin, 384 (1939).

<sup>3)</sup> Conf.: E. ROSENBOHM and F. M. JAEGER, Proc. Kon. Akad. v. Wetensch., Amsterdam, **39**, 374 (1936); etc.

galvanometer with photographic recording, — the metal being investigated in a high-vacuum furnace with electrical heating <sup>1)</sup> and the measurements being executed by the aid of a precision THOMSON-bridge with 5 decades and a normal standard-resistance of 0,01 Ohm. The way in which the bar of *ruthenium* was fixed between the leads, was as follows: as the metal was too hard to drill a hole in the terminal faces, these holes were provided for by slowly grinding them conically out during many days by means of carborundum. Then two equally conically ground platinum wires of 2 m.M. thickness were tightly fitted in these holes; these wires, in their turn, were linked to platinum rods of 4 m.M. thickness which, — outside the zone of high temperatures, — were connected with two corresponding copper-bars. The resistance of those leads at different temperatures was, of course, afterwards separately determined when the measurements were finished and then the corresponding corrections were applied. The results thus obtained are recorded in Table I and graphically represented in Fig. 1.

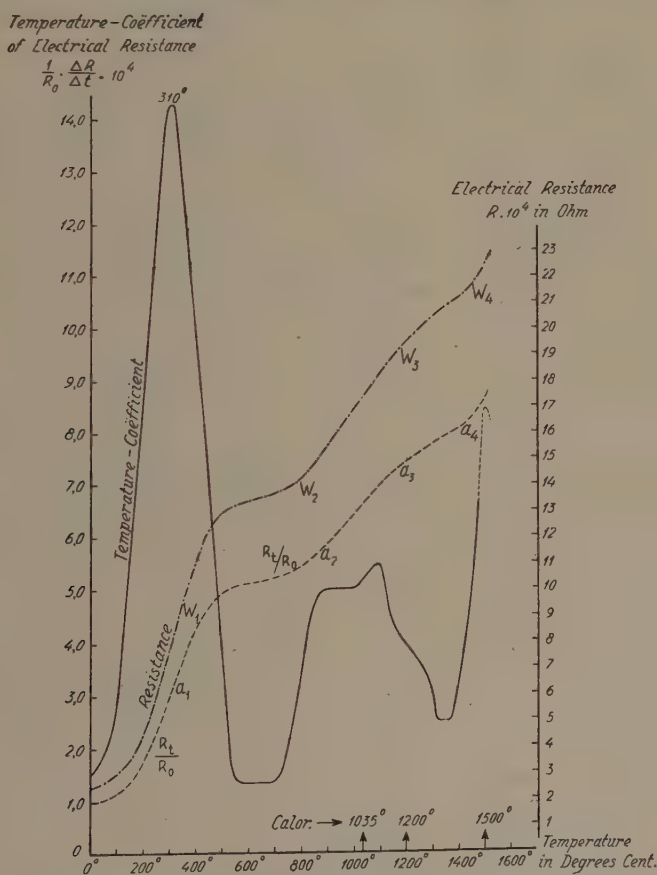


Fig. 1. The Electrical Resistance and its Temperature-Coefficient in the Case of Ruthenium between 0° and 1500° C.

<sup>1)</sup> For the special alterations applied to the original equipment, see § 3.

TABLE I. Electrical Resistance of sintered, pure Ruthenium between 0° and 1475° C.

Temp. $t$ in °C.:	$R_t \cdot 10^4$ in Ohm:	$\frac{1}{R_0} \cdot \frac{\Delta R}{\Delta t} \cdot 10^4$ :	$R_t/R_0$ :	Temp. $t$ in °C.:	$R_t \cdot 10^4$ in Ohm:	$\frac{1}{R_0} \cdot \frac{\Delta R}{\Delta t} \cdot 10^4$ :	$R_t/R_0$ :
0°	(2.62)	—	1.00	775°	14.12	2.67	5.39
25	2.71	1.67	1.035	800	14.32	3.42	5.47
50	2.80	1.83	1.069	825	14.58	4.27	5.56
75	2.93	2.29	1.119	850	14.88	4.73	5.68
100	3.10	3.05	1.184	875	15.20	4.96	5.80
125	3.34	4.12	1.275	900	15.53	5.04	5.93
150	3.66	5.50	1.397	925	15.86	5.04	6.05
175	4.07	7.18	1.554	950	16.19	5.04	6.18
200	4.58	9.01	1.749	975	16.52	5.04	6.30
225	5.23	10.68	1.997	1000	16.85	5.04	6.43
250	6.01	12.37	2.294	1025	17.18	5.11	6.56
275	6.85	13.74	2.634	1050	17.52	5.27	6.69
300	7.77	14.20	2.967	1075	17.87	5.42	6.82
325	8.71	13.90	3.327	1100	18.23	5.49	6.96
350	9.57	12.83	3.653	1125	18.59	4.96	7.09
375	10.37	11.45	3.960	1150	18.88	4.27	7.21
400	11.06	10.08	4.22	1175	19.16	4.09	7.31
425	11.71	8.48	4.47	1200	19.41	3.97	7.41
450	12.23	7.18	4.67	1225	19.66	3.74	7.50
475	12.64	5.34	4.82	1250	20.01	3.56	7.64
500	12.94	3.51	4.94	1275	20.24	3.42	7.73
525	13.12	2.14	5.01	1300	20.46	3.13	7.81
550	13.21	1.37 <sup>5</sup>	5.04	1325	20.65	2.90	7.88
575	13.30	1.37 <sup>5</sup>	5.08	1350	20.84	2.90	7.96
600	13.39	1.37 <sup>5</sup>	5.11	1375	21.03	2.90	8.03
625	13.48	1.37 <sup>5</sup>	5.15	1400	21.22	3.05	8.10
650	13.57	1.37 <sup>5</sup>	5.18	1425	21.44	3.82	8.19
675	13.66	1.37 <sup>5</sup>	5.21	1450	21.73	5.19	8.30
700	13.75	1.37 <sup>5</sup>	5.25	1475	22.12	6.64	8.44
725	13.84	1.53	5.28	1500	—	—	—
750	13.96	2.04	5.33				

Length of the bar = 0.048 M; cross-section 13 m.M<sup>2</sup>.

$R_0$  = approx.  $7.10 \cdot 10^{-6} \Omega$ . The resistances are corrected for the resistance of the leads at the corresponding temperatures.



As far as the poorly defined state of the *Ru*-bar allows of drawing conclusions with any degree of certainty, it at least appears evident that truly several changes of state occur in the metal. As to the peak at 310° C. it seems problematic whether this point does corresponds to a transition-

temperature in the ordinary sense of the word or not. The change here observed reminds somewhat of that, met with in the case of electrolytical iron between 150°—200° C. which, — as was previously stated, — does certainly not correspond to a change of the internal structure of the metal<sup>1)</sup>. Between about 540° C. and 840° C. the curve of  $\frac{dR}{dT}$ , after a short interval of constancy, rapidly rises and then reaches a peak between 1050°—1100° C.; subsequently, however, it slopes down to a deep minimum, — this branch of the curve manifesting a conspicuous inflection-point at 1200° C.; subsequently it once more very steeply rises to a probable maximum in the vicinity of 1500° C. Although no high degree of accuracy can be attributed to the temperatures here indicated, we may safely conclude from these data, that the behaviour is in general outlines in agree-

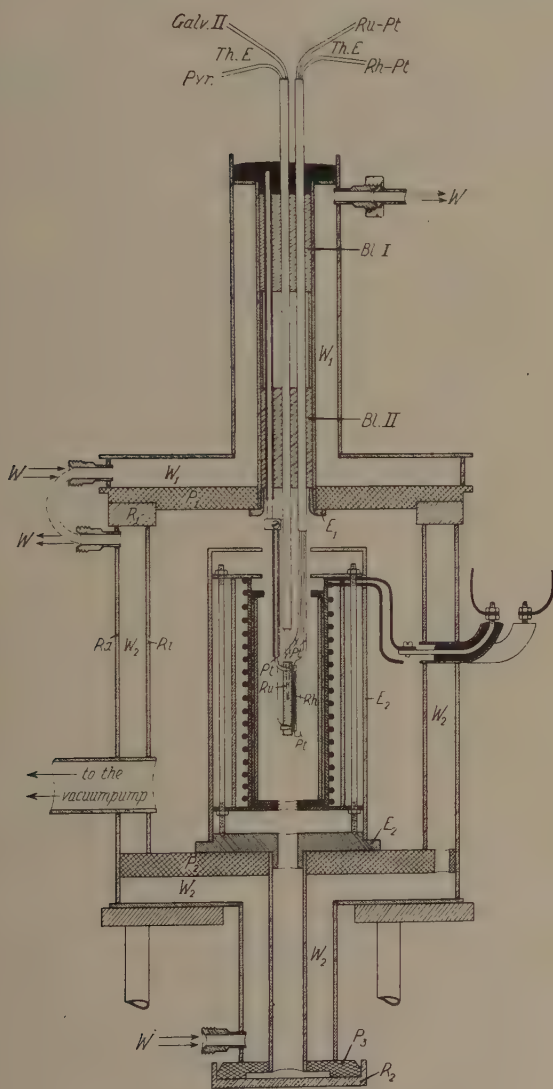


Fig. 2. The Heating-apparatus used, with Experimental Device.

ment with the results obtained in our former calorimetrical investigations<sup>2)</sup>; so that the data here collected really seem sufficiently to corroborate our

<sup>1)</sup> F. M. JAEGER, E. ROSENBOHM and J. A. ZUITHOFF, Recueil d. Trav. d. Chim. d. Pays-Bas, **57**, 1323, 1327—1336 (1938).

<sup>2)</sup> F. M. JAEGER and E. ROSENBOHM, ibidem, **51**, 32, 45, (1932).

previous conclusions concerning the existence of *several different states* of metallic *ruthenium*.

§ 3. The determinations concerning the thermoelectrical properties of a thermocouple consisting of *ruthenium* and *platinum* led to the same conclusions.

These measurements again presented great difficulties as a consequence of the physical properties of the metal and of the restricted dimensions of the metallic bar available. As *ruthenium* cannot be drawn into wires, the usual method of determining thermoelectrical forces  $E$  cannot be applied; but on the other hand, because we for our purpose need not so much this thermoelectrical force itself as well its temperature-coefficient  $\frac{dE}{dT}$ , — for

attaining the end in view it yet proved possible to use the same *ruthenium*-bar of 5 c.m. length as in our former experiments, if only the following supplementary experimental trick were applied. A thin platinum-wire (0,2 m.M.) was inserted into the conically-shaped hole in the lower terminal end of the bar (§ 2) by pressing it into this hole by means of a short, exactly fitting conical platinum rod; the same was done at the upper end of the bar, but this time a *long* platinum rod of 4 m.M. thickness was used. The upper end of the latter was connected with the water-cooled, disk-shaped bottom  $P_1$  of the vacuum-vessel formerly used in the case of the measurements of the thermal expansion-coefficients of metals <sup>1)</sup>. In stead of the previously applied <sup>2)</sup>, evacuated tube of Pythagoras-mass placed in the interior of the platinum resistance-furnace, we used the arrangement represented in Fig. 2. It consists of a double-walled, water-cooled and evacuated steel-cylinder, in the interior of which the electrical furnace itself is mounted. The steel cylinder is at its upper end hermetically closed by means of a double-walled and water-cooled cover, provided with a conically ground rim; the latter part can, when necessary, easily be removed and exchanged by another one, which is more effectively adapted to other special experimental devices. The thick platinum rod mentioned in the above is simultaneously used as a support for the ruthenium-bar; so that in combination with the thin platinum wire described, a differential thermocouple:  $Pt—Ru—Pt$  is formed; the upper part of this will in the heated furnace always be at a somewhat *lower* temperature than the junction at the lower end, because of the faster heat-abduction through the thick platinum rod. In this way there will always exist a certain potential-difference  $\Delta E$  between the two ends, the value of which at each temperature can photographically be recorded in the usual way <sup>3)</sup> by means of the twin-galvanometer-equipment.

<sup>1)</sup> E. ROSENBOHM, *Physica*, 5, 385 (1938).

<sup>2)</sup> E. ROSENBOHM and F. M. JAEGER, Proc. Kon. Akad. v. Wetensch., Amsterdam, 39, 366 (1936).

<sup>3)</sup> Ibidem, p. 469.

Now, if the difference in temperature at the two terminals of the *ruthenium*-bar during the gradual heating of the furnace would only remain constant, the observed values of  $\Delta E$  at each temperature would immediately furnish those of the temperature-coefficients  $\frac{\Delta E}{\Delta T}$  desired. But evidently this is not the case: when the temperature of the furnace is slowly increased, the difference of the temperature at the two terminals of the bar will also gradually be augmented; and as a consequence of this,  $\Delta E$  will ever more be extended over increasing ranges of the temperature and thus the photographically recorded curve for  $\frac{\Delta E}{\Delta T}$  will represent a *distorted* image of the true one. To eliminate the said distortion, the two junctions of another differential thermo-element of *Pt—PtRh—Pt* now were also connected with the terminals of the *ruthenium*-bar and its indications read-off by means of a third, suitably arranged galvanometer. In this way the temperature-differences  $\Delta E'$  (in microvolts) between the two ends of the

bar could be measured at each moment and the mean temperature of the bar itself could simultaneously be determined in such a way, that during each determination of  $\Delta E'$ , the light-source of the photographic recording-apparatus was switched-off during ten seconds, so that the short interruptions in the recorded curve thus obtained, by their abscissae, — which were read-off by means of the third galvanometer, — immediately indicated the corresponding mean temperatures of the bar.

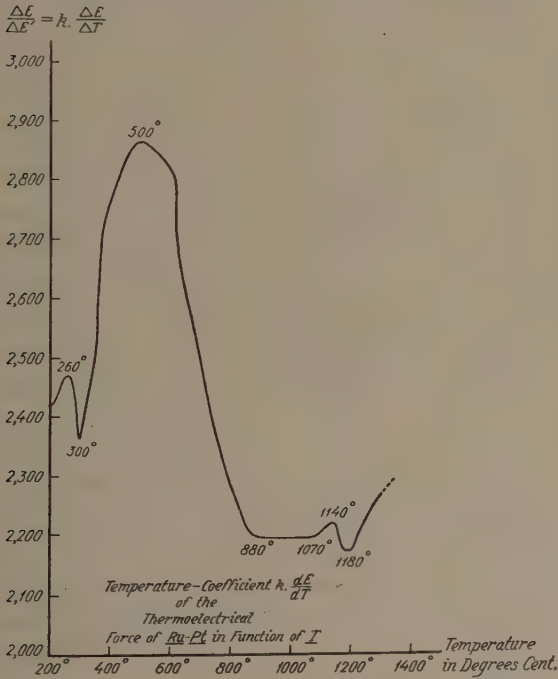


Fig. 3. The approximate Temperature-Coefficients of the Thermoelectrical Force of Ruthenium against Platinum between 200° and 1300° C.

now will represent the sought temperature-coefficients of the thermocouple: *Ru—Pt*, the temperatures of which now are expressed in microvolts in stead of in degrees Centigrade: more exactly, these differentials give the temperature-coefficients of the thermocouple: *Ru—Pt* in comparison with

The differentials  $\frac{\Delta E}{\Delta E'}$



those of the *Pt—PtRh*-thermocouple, because the integral thermoelectric force of the latter is *not* an exactly *linear* function of *T*, but will graphically be represented by a feebly curved line. Because of the fact, however, that the shape of the curve  $\frac{\Delta E}{\Delta E'}$  can not appreciably differ from that for  $\frac{dE}{dT}$  and because absolute values of this coefficient can neither be obtained in this way, nor are they of direct interest for our purpose, — we here made no further attempts to determine the still remaining slight corrections, e.g. by a special calibration of the *Pt—PtRh—Pt*-thermocouple. This is excusable, because the lack of homogeneity of the *ruthenium*-bar, — the density of which was appreciably lower than that of the molten and

TABLE II. Approximate Values of $k \frac{dE}{dT}$ (resp. $\frac{dE}{dE'}$ ) of pure, sintered Ruthenium against Platinum, between 200° and 1300° C.					
Mean Temperature <i>T</i> in Degrees Cent.:	$\frac{dE}{dE'}$ in M.V.:	Mean Temperature <i>T</i> in Degrees Cent.:	$\frac{dE}{dE'}$ in M.V.:	Mean Temperature <i>T</i> in Degrees Cent.:	$\frac{dE}{dE'}$ in M.V.:
200°	2.424	520°	2.855	840°	2.232
220	2.446	540	2.849	860	2.206
240	2.463	560	2.840	880	2.198
260	2.471	580	2.830	900	2.198
280	2.445	600	2.818	1000	2.198
300	2.360	610	2.778	1060	2.198
320	2.424	614	2.690	1080	2.202
340	2.482	616	2.678	1100	2.207
360	2.570	620	2.663	1120	2.213
362	2.667	640	2.622	1140	2.220
380	2.716	660	2.580	1150	2.206
395	2.760	680	2.538	1160	2.182
398	2.757	700	2.488	1180	2.172
400	2.750	720	2.436	1200	2.179
420	2.782	740	2.391	1220	2.197
440	2.820	760	2.350	1240	2.214
460	2.850	780	2.316	1260	2.231
480	2.861	800	2.275	1280	2.250
500	2.862	820	2.251	1300	2.267

solidified metal, — does not furnish any guarantee as to the real significance of such "absolute" values, but, on the contrary, makes the latter appear as more or less illusory data.

In the table II the values of  $\frac{\Delta E}{\Delta T}$  at the corresponding mean temperatures  $T$ , have been collected; whilst in Fig. 3 they are graphically represented as an approximate substitute for  $\frac{\Delta E}{\Delta T}$  versus the temperature;  $\frac{\Delta E}{\Delta T}$  (appr.) is here expressed in microvolts and  $T$  in degrees Centigrade. The scale of the graph chosen is the same as that of Fig. 1; so that the two figures can immediately be compared.

The true temperature-difference between the two ends of the *ruthenium*-bar studied are at the highest temperatures about  $10^\circ \text{C.}$ ; the direction of the current in the *Ru*—*Pt*-couple appears to be the same as in the thermocouple: *Pt*—*PtRh*.

On comparing the curve of Fig. 3 with that of Fig. 1, it becomes clear that, *in general outlines*, the elevations and depressions in the curve of Fig. 3 appear up to about  $1050^\circ \text{C.}$  in mirror-symmetrical situations with respect to those in Fig. 1; but at higher temperatures the curve loses this character.

The second part of the curve (between about  $1070^\circ$  and  $1200^\circ \text{C.}$ ) is rather more analogous to that of Fig. 1, with the exception that the inflection-point at  $1200^\circ \text{C.}$  of Fig. 1 is here replaced by a branch going upwards till  $1300^\circ \text{C.}$  The minimum at  $300^\circ \text{C.}$  in Fig. 3 evidently corresponds to the maximum at  $310^\circ$  in Fig. 1; the maximum at  $260^\circ \text{C.}$  in Fig. 3 has no corresponding minimum in Fig. 1, but seems to be co-ordinated with the rapidly rising values of the resistance  $W$  in Fig. 1 which start in the vicinity of  $220^\circ \text{C.}$ ; whilst the horizontal part in Fig. 3 between  $880^\circ$  and  $1070^\circ$  corresponds to the similar part in the curve of Fig. 1 between  $880^\circ$  and  $1040^\circ \text{C.}$

§ 4. Finally we again have made some tentatives to localize the most important transitions-points in the differential heat-capacity-curve of *ruthenium* by direct comparison with the heat-capacity of pure molybdenum after the usual method of photographic recording with the twin-galvanometer of SALADIN—LE CHATELIER <sup>1)</sup>. The experiments were executed in the way previously indicated, but with the furnace-equipment described in the present paper. The results are graphically represented in Fig. 4.

It appears that the curve for the *differential* heat-capacity of the two metals again shows a number of discontinuities: at  $312^\circ \text{C.}$ , at about  $1040^\circ \text{C.}$ , at  $1200^\circ \text{C.}$ , etc. Previously <sup>2)</sup>, we found for the transition-

<sup>1)</sup> Conf.: E. ROSENBOHM and F. M. JAEGER, Proc. Kon. Akad. v. Wetensch., Amsterdam, **39**, 366 (1936).

<sup>2)</sup> F. M. JAEGER and E. ROSENBOHM, Rec. d. Trav. d. Chim. d. Pays-Bas, **51**, 35 (1932).

temperature at  $\alpha \rightleftharpoons \beta$ -ruthenium <sup>1)</sup>: 1035° C.; for that of  $\beta \rightleftharpoons \gamma$ -ruthenium: 1190° C., whilst the transformation-point of  $\gamma \rightleftharpoons \delta$ -ruthenium was determined at about 1500° C. There only remains the conspicuous maximum at

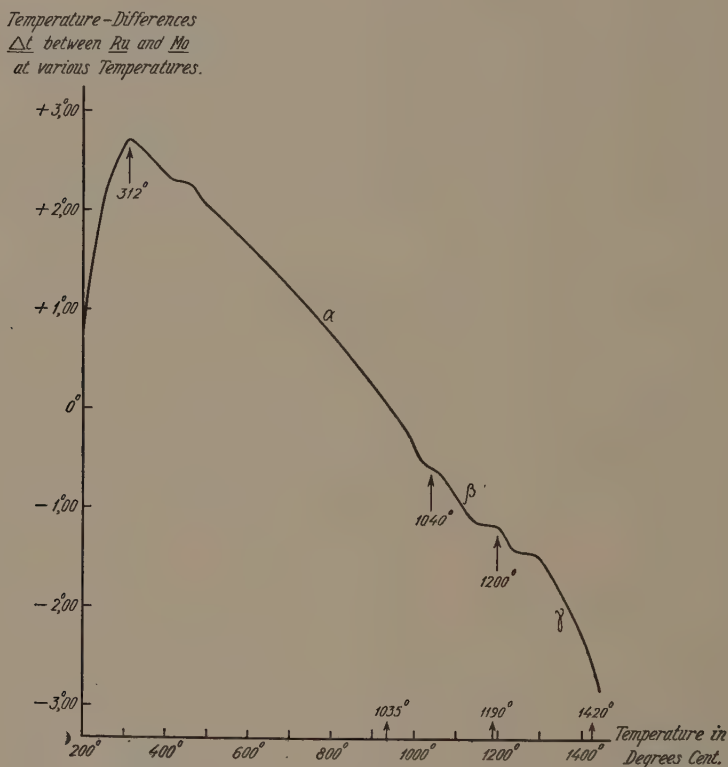


Fig. 4. The differential Heat-capacity-Curve between Ruthenium and Molybdenum at various Temperatures.

301°—312° C. If this point might be considered as a true “transition”-temperature, *ruthenium* would be *pentamorphous* in stead of *tetramorphous*. But we have no certainty of this: the shape of the curve at this temperature has quite another character than at the other transformation-temperatures and the curve has a different course reminding one of the analogous one in the case of *iron* between 150° and 200° C. previously stated by us, as well as by a number of other authors. At the moment by some of them it is attributed to another cause <sup>2)</sup>. But as a whole, we can deduce from these supplementary investigations, that our former conclusions as to the multiple allotropic changes of the metal, are *correct*; so that the analogy between the behaviour of *ruthenium* and *iron* now appears, indeed, firmly to be established.

<sup>1)</sup> The *ruthenium* there used, consisted of small, homogeneous *globules*.

<sup>2)</sup> F. M. JAEGER, E. ROSENBOHM and A. J. ZUITHOFF, *Recueil*, **57**, 1327, 1336 (1938).



**Medicine.** — *Position nystagmus caused by "Neck-reflexes", especially in cases of arachnoiditis posterior.* By Prof. A. DE KLEYN.

(Communicated at the meeting of January 25, 1941.)

Posterior arachnoiditis has for the last years occupied a prominent place in the interest of neurologists and neuro-surgeons. This can easily be understood if one remembers that in many cases of posterior arachnoiditis a diagnosis of serious cerebellar disease is made. Operation, performed in these cases, reveals an affection which appears to be of a rather harmless character. As this arachnoiditis also develops after an otitis media, otologists are also interested in this problem of modern neuro-surgery.

In 1932 BROUWER <sup>1)</sup>, in a clinical lesson, exposed again that usually it is impossible to diagnose a posterior arachnoiditis by means of the ordinary neurological appliances. Ventriculography, which on occasion enables to make the correct diagnosis, is, however, attended by many difficulties and risks. So it has become desirable to dispose of new clinical symptoms enabling to diagnose an arachnoiditis with more certainty and without the aid of ventriculography. BROUWER himself pointed to the varying character of the symptoms in arachnoiditis with its ups and downs, especially present in the first phase of the disease, and failing in cases of cerebellar tumors with their slow progressive course. In this last disease symptoms of the medulla might be more prominent than in cases of arachnoiditis. Especially absence of nystagmus in serious cerebellar syndromes (as writes BROUWER) must always draw our attention in this direction.

I myself was able to observe some cases of posterior arachnoiditis in which an uncommon form of position-nystagmus was present. This position-nystagmus caused by so-called "Neck-reflexes" may perhaps also be of diagnostic value.

Position nystagmus is taken to be a nystagmus appearing when the head is in a certain position. There are different forms.

I. The first cause may be a certain position of the head in space. The nystagmus, developing under such circumstances, is considered to be of labyrinthine origine; ROTHFELD <sup>2)</sup> found it in rabbits which were poisoned with alcohol. Many investigators were able to observe this form of nystagmus also in man.

II. Another form of nystagmus develops when the position of the head is changed in relation to the trunk, i.e. on turning the head back and

<sup>1)</sup> B. BROUWER, Ned. tijdschr. v. Geneeskunde 76, 223 (1932).

<sup>2)</sup> J. ROTHFELD, Arb. a. d. Neurol. Inst. d. Wiener Universität 22, 88 (1913).

simultaneously into one of the lateral positions <sup>1)</sup>). In turning f.i. the head to the right lateral position the left vertebral artery is obstructed, by which the circulatory relations in the labyrinth and in the vestibular region become altered. The circumstances leading to such a nystagmus have been published elsewhere <sup>1)</sup>).

III. The third, up till now only but rarely examined form, necessitates a certain lateral position of the head for some time, after which it is turned into the opposite lateral position <sup>2)</sup>). If, however, the head is turned from the dorsal into a lateral position, no nystagmus develops.

IV. The fourth of the known types of nystagmus can be elicited by the mere turning of the head in relation to the trunk. This last form of nystagmus is the subject of this paper and observed in some cases of posterior arachnoiditis.

In our first communication <sup>3)</sup> we called this form: position nystagmus caused by "Neck-reflexes". The term neck-reflexes must be taken in a general sense and not completely identified with the tonic neck-reflexes on extremities and eyes, which are conducted via the cervical roots. It seems not by any means improbable to me that the position nystagmus which, in cases of posterior arachnoiditis, can be produced by turning the neck, is due to disturbances in the liquor circulation, which may easily develop under the above mentioned pathological circumstances.

V. BIEMOND <sup>4)</sup> proved, by experimental and clinical investigation, that also stimulation of the cervical roots may produce a position nystagmus. He was also able, by anatomical investigation, to indicate the presence of a connection between the cervical roots and the vestibular region.

Therefore this fifth form of nystagmus must be added to the four known up till now.

BÁRÁNY <sup>5)</sup> considered the term "Neck-reflexes" to be wrong as, according to his opinion, this form of position nystagmus was always due to disturbances in the circulation of the neck vessels, caused by turning of the neck. However, for our cases, we could not confirm this idea: pressure on the neck vessels, either on the carotid artery or on the jugular vein, caused no nystagmus. It appears, however, from BÁRÁNY's communication, that especially in arteriosclerosis the occurrence of nystagmus is sometimes due to the above mentioned facts.

BORRIES <sup>6)</sup> made another objection against our explanation of the nys-

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<sup>1)</sup> A. DE KLEYN und NIEUWENHUYSE, *Acta Oto-Laryng.* **11**, 155 (1927). A. DE KLEYN und C. VERSTEEGH, *Ztschr. f. Nervenheilk.* **132**, 157 (1933).

<sup>2)</sup> A. DE KLEYN, *Confinia Neurologica* **2**, 287 (1940).

<sup>3)</sup> A. DE KLEYN und C. VERSTEEGH, *Acta Oto-Laryng.* **6**, 99 (1924). A. de KLEYN und C. VERSTEEGH, *Dtsch. Ztschr. f. Nervenheilk.* **132**, 157 (1933).

<sup>4)</sup> A. BIEMOND, *Proc. Kon. Ned. Akad. v. Wetensch.*, Amsterdam **42**, 370 (1939) en **43**, 3 (1940).

<sup>5)</sup> R. BÁRÁNY, *Acta Oto-Laryng.* **7**, 1 (1925).

<sup>6)</sup> G. BORRIES, *Arch. f. Ohrenheilk.* **112**, 53 (1924).

tagmus in our first case. At the time it was only stated by us that the nystagmus developed when the patient, lying in the dorsal position, turned her head to the left lateral position, or when lying completely in the left lateral position, the trunk was brought in the recumbent and the head was left in the left lateral position. BORRIES thought that the special, i.e. lateral position of the head might play a role in the development of the position nystagmus. Both the turning of the head in relation to the trunk and the labyrinthine factor, due to the lateral position of the head, should be necessary to cause the stimulation by which the position nystagmus appeared. BORRIES' objection, however, also appeared to be wrong for our case. The nystagmus also develops when, with the patient in sitting position, the head was turned in the horizontal plane to the left and right side. However, in this way, no tonic labyrinthine reflexes can be elicited, as this alteration of position leaves the position of the labyrinths to the horizontal plane unchanged.

My cases of posterior arachnoiditis, showing the above mentioned form of position nystagmus, are recorded below.

1. Miss J., 34 years.

Since 1926 patient was complaining of slight attacks of head ache. At the same time she was suffering from a kidney disease. After some smaller operations which had no effect, a hydronephrosis caused by vessel pressure, necessitated a left-sided kidney-extirpation in March 1931. It appeared that the mother had the same kidney affection. In May 1931 the first attack of severe dizziness developed. The environs went up and down; when the attack was over she vomited. The attacks reoccurred in July, September and October and it was stated they started a few days before menstruation. Patient was then examined by us.

Examination of the patient on October 27th, 1931, in an attack-free period, revealed the following.

*Acoustic function:* At right a slight labyrinthine deafness was present, the whispering voice, however, was heard at 5 M. Left side completely normal.

*Vestibular function:* No different forms of spontaneous nystagmus, no past pointing etc.

*Caloric examination:* both labyrinths were normally stimulated by cold and warm water. Reactive past pointing: no abnormality.

Hereafter the attacks occurred each month just before or between two menstruations. Administration of corpus luteumpreparations had no effect. The head ache grew worse during the attacks, she also complained of diplopia. The patient was lying in dorsal position with the head bent backwards or in the right lateral position. Turning of the head to the left lateral position could not be supported as dizziness and vomiting followed immediately.

On April 1st 1932, patient was admitted to the hospital. She had an attack with severe ache in the back part of the head. She was lying in bed in the dorsal position, the head bent backwards. She now showed a completely different syndrome as on examination in the attack-free period. In dorsal position, looking straight forward, no nystagmus was present; looking to the right elicited a slight right-sided horizontal rotatory nystagmus, looking to the left a nystagmus to the left of the same character. Other regards revealed no nystagmus. However, on turning the head to the left lateral position, a vertical nystagmus followed by a rotatory nystagmus to the left developed, accompanied by vertigo and vomiting. Turning of the head to the right lateral position did not cause neither dizziness nor nystagmus, just as when the patient was in the left lateral position (with the head in the same position as the trunk). When, however, the head was kept



in this position and the trunk changed to the recumbent position, a vertical nystagmus together with vertigo and vomiting appeared, followed by a nystagmus to the left. Pressure on the neck vessels caused no nystagmus.

Summarizing, this patient showed a distinct position nystagmus dependent on the position of the head in relation to the trunk. The nystagmus belonged to the above mentioned fourth form:

- I. Patient in dorsal position: no nystagmus.
- II. Trunk in dorsal position, head in left lateral position: vertical nystagmus upwards, followed by rotatory nystagmus to the left.
- III. Trunk in dorsal position, head in right lateral position: no nystagmus.
- IV. Patient completely in left lateral position: no nystagmus.
- V. Head in left lateral position, but trunk in dorsal position: vertical nystagmus upwards followed by a rotatory nystagmus to the left.

Further vestibular examination revealed no abnormalities. Just as in the healthy period the caloric stimulation of the labyrinths with cold and warm water, was normal. Horizontal, vertical and rotatory nystagmus, after turning to the right and to the left, were equal and of normal duration.

Extensive internal examination: no abnormalities, urine normal.

Neurological examination (Dr. STENVERS): Fundi no abnormalities, visual field on both sides normal. Corneal reflex at right: positive; at left negative; sometimes negative on both sides. The optokinetic nystagmus in all directions normal. Abdominal reflexes weak but equal. The sensation in both legs was sometimes badly recorded. Speech was slurring as if the patient had an apple in her mouth. No further neurological symptoms. In looking upward a vertical nystagmus, directed upward, sometimes appeared, in this case the patient also complained of diplopia.

Röntgenological examination: X-rays of the skull and of the petrous bone showed no abnormalities.

The inference of the neurologist was: A process of the fourth ventricle, not yet causing papilloedema, is thought probable, together with an obstruction of the aqueductus which, as it seems, may be neutralized by a certain position of the head.

On April 18th a trepanation was performed (Dr. WALLER) at which the cerebellum was exposed. An extensive posterior arachnoiditis was found. Beneath the dura many membranes were found on the cerebellum, with accumulation of cerebrospinal fluid. The membranes were opened and partly removed.

Patient had to be operated upon several times before she was without complaints. The above mentioned position nystagmus, however, could no more be elicited after the first operation.

## II. v. O., male, 42 years, Chauffeur.

Five months previously, creeping from under his car, he suddenly had the sensation of getting a slap on his head, after which he developed a severe head ache, creeping to his neck. He now also complains chiefly of pain in the neck and in the back of the head. This pain was the worst at morning and diminished in the evening. He also suffered from tinnitus, especially of the left ear.

It seemed he had before also complained of head ache. Four years previously his physician referred him to an ophthalmologist, who prescribed spectacles, after which the complaints disappeared. Since four months his gait has become unsteady, swinging as though he were drunk, mostly diverging to the right. He also became dizzy if his head ache grew worse. His wife told his gait was already disturbed for the last half year. Since his illness he talked as if he had his mouth full. The last four weeks he also vomited frequently.

The family history revealed the occurrence of tuberculosis, especially from maternal side. Furthermore the patient stated spontaneously that he was especially sick and dizzy when, lying in the dorsal position, he turned his head to the left. No dizziness, sometimes

vomiting, developed when he changed completely from the dorsal to the left lateral position. Lying in the right lateral position or turning his head to the right when in the dorsal position did not cause neither vertigo nor vomiting.

Examination on July 26th, 1933:

*Acoustic function:* Whispering voice was heard on both sides at normal distances. The ear drums showed no abnormalities.

*Vestibular function:* Only a slight horizontal nystagmus developed spontaneously after looking extremely to the right or to the left. No spontaneous past pointing in the various planes.

On looking straight forward the following was noted:

- I. Trunk and head in dorsal position: no nystagmus.
- II. Trunk in dorsal position, head in right lateral position: no nystagmus.
- III. Trunk in dorsal position, head in left lateral position: rotatory nystagmus to the left, patient developed also vomiting.
- IV. Completely in left lateral position (head in symmetric position with the trunk): no nystagmus<sup>1)</sup>.
- V. Head in left lateral position and trunk changed into the dorsal position: severe rotatory nystagmus to the left developed immediately.

A caloric reaction of both labyrinths after syringing the ears with cold and warm water was present, simultaneously a typical reactive past pointing developed. It also appeared that a so-called "Nystagmusbereitschaft" for a nystagmus with its quick component to the left, was present. That means that if a nystagmus was elicited with its quick component to the left, this nystagmus was always stronger than that elicited with its quick component to the right. After syringing both sides with 5 cc. of warm water (35°) a definite nystagmus only developed at the right side; syringing with 5 cc. of warm water (45°): the nystagmus only appeared at the left. By syringing simultaneously both ears (75 cc. of 19°), no nystagmus could be elicited.

*Internal examination:* No abnormalities. Blood pressure, blood picture and urine were normal. Reactions of WASSERMANN, SACHS GEORGI and KAHN negative.

Examination of the cerebrospinal fluid gave the following result: Pandy +, Nonne —, cells  $\frac{2}{3}$ .

*Neurological examination* (Dr. STENVERS): Cerebral nerves no abnormalities, no choked discs. Reflexes normal. Diadochokinesia and sensation intact. Patient talked as if he had his mouth full. If he had head ache it sometimes appeared that in looking upward a nystagmus was present with its quick component upward. Eyemovements intact.

Röntgenological examination of the cervical vertebrae proved that the intervertebral disk between C II and C III was not distinctly seen, which corresponded to a tender hard swelling present at C III.

Thus, this patient has the same form of position nystagmus as our last patient. In both cases the diagnosis could be made, almost without examination, from the typical anamnesis.

Besides, the second patient showed a slight nystagmus being in complete lateral position, during a repeated examination. In this case a „Nystagmusbereitschaft" was present, in which by syringing both ears simultaneously, no nystagmus was produced. This could be due to the fact that a process or pressure, situated orally<sup>2)</sup> of the vestibular nucleus area<sup>3)</sup>, was present.

<sup>1)</sup> Examination, performed after a few days, revealed a rotatory nystagmus to the left, but weaker than that described under III.

<sup>2)</sup> A. DE KLEYN und C. VERSTEEGH, D. Ztschr. f. Nervenheilk. 132, 157 (1933).

<sup>3)</sup> Probably by pressure of the liquor in the aqueduct or in the ventricles, as later a posterior arachnoiditis was found.

The neurologist doubted whether a diagnosis of arachnoiditis posterior or a process in the cerebellar or cysticereal vermis had to be made.

Trepanation (performed by Dr. OLJENICK) revealed a posterior arachnoiditis; both cerebellar tonsils were extremely fixed and adhered beneath the atlas.

The patient was re-operated, but then left for a foreign country. However, he informed us his complaints had vanished. Exceptional circumstances made it impossible for me to re-examine the patient after the operation.

The symptoms of the next case obliged the neurologist to made the probable diagnosis of posterior arachnoiditis. Spontaneous recovery made an operation unnecessary; therefore the diagnosis could not be verified.

**Patient H.**, 30 years, green-grocer.

For 25 years patient had discharge of the left ear. On occasion he had pain-attacks, which disappeared after poulticing the ear, which caused excretion of blood and pus. Last years he also had a smarting pain behind the ear, which lasted night and day. However, he could still do his work. He did not vomite. As the attacks of head ache became progressively worse he applied to an otologist who performed a radical-operation on May 8th 1940. Previous to this operation it had been shown that normal vestibular reactions could be elicited at the diseased side. Röntgenological examination had revealed a cavity in the mastoid. Consequently a cholesteatoma was found at the operation.

Hereafter the head ache disappeared and patient was discharged on May 17th 1940, apparently recovered, but on May 25th he complained again of head ache and even of diplopia. The complaints became worse and on June 4th the ophahalmologist found a bilateral papilloedema of  $\pm 5$  dioptria.

Temperature was normal, pulse about 60 per minute. X-rays showed no disturbances of the skull or the petrous bone. Patient was not sick or dizzy. No aphasia was present, the reflexes were normal, nystagmus was absent. Speech was somewhat slurring, but his friend stated this had always been the case. In view of his apparently serious condition patient was admitted to the department of Professor BROUWER.

Here the following facts were recorded:

Distinct papilloedema were found with small hemorrhages in the fundi. No cerebellar symptoms. Also here no disturbances were seen on the X-ray of the skull. The blood picture and the sedimentation rate were normal. A lumbar puncture revealed the presence of a pressure of 360 mm. After Queckenstedt's test a small increase at the left appeared, at the right the increase proved to be more pronounced (to 440 mm). The liquor was colourless and clear. The reaction of Nonne was negative, Pandy positive, cells  $\frac{3}{3}$ . The reactions of WASSERMANN, SACHS GEORGI and LANGE were negative. The reaction of PIRQUET, both for human and bovine tuberculine, was positive.

On extensive neurological examination no disturbances, except a slight right-sided abducens paresis, were found. The right ear drum was dull and had a small perforation just before the handle of the hammer. At the left the middle ear cavity still seemed to secern. Rhinological and laryngological examination: no disturbances.

*Acoustic function:* at right the whispering voice was heard at more than 6 M, the tuning fork test was normal. At left the auditory function was decreased to the ordinary voice ad concham; the tuning fork test revealed an affection of the middle ear. On WEBER's test patient localized to the diseased left side.

*Vestibular function:* no spontaneous nystagmus developed by looking in different directions, no spontaneous past pointing was present. Examination of rotatory nystagmus revealed that the horizontal, vertical and rotatory types of nystagmus were present after turning to the right and to the left and of equal and normal duration.



The caloric reaction of both labyrinths by syringing the ears with cold and warm water, was present.

The Kip-reactions were normal around the longitudinal and bitemporal axis.

*Position nystagmus:*

- I. Patient completely in dorsal position: no nystagmus.
- II. Patient completely in right lateral position: no nystagmus.
- III. Patient completely in left lateral position: no nystagmus.
- IV. Patient in dorsal position, head in right lateral position: nystagmus to the left, scarce small movements<sup>1)</sup>.
- V. Patient in dorsal position, head in left lateral position: nystagmus horizontal to the right, distinct movements.

Optokinetic nystagmus: examination, both of the so-called subcortical and cortical form, revealed no disturbances.

Patient was discharged from the neurological department on June 28th, when the head ache and diplopia had disappeared and the left fundus had become normal; the right fundus still showed a small swelling. Examination on position nystagmus revealed the remarkable fact that this nystagmus had changed its direction when the patient was in the right lateral position, whereas, with patient in the left lateral position, the nystagmus was no more present.

- I. Patient in dorsal position: no nystagmus.
- II. Patient completely in right of left dorsal position: no nystagmus.
- III. Patient in dorsal position, head in left lateral position: no nystagmus.
- IV. Patient in dorsal position, head in right lateral position: horizontal nystagmus to the right (see under IV mentioned above: horizontal nystagmus to the left).
- V. Head in right lateral position and trunk turned in dorsal position: horizontal nystagmus to the right.

Later informations revealed that the patient made a complete recovery.

Although in this case no operation was performed, the otitis and the observed symptoms made the diagnosis posterior arachnoiditis very probable.

In the next case the diagnosis posterior arachnoiditis is, contrary to the preceding case, very doubtful. Extensive otologic examination of the patient only could be performed but once. However, the patient had the same type of position nystagmus as described in our other patients.

**Patient S., 51 years. Saddler.**

Three years previously patient was examined at the out-patient department as he was suffering from a left-sided otitis media. He did not come back, prevented to walk by an accident. However, he informed us he had had no complaints until a week before examination when dizziness developed. The surroundings seemed to turn to the left, he himself feeling as if he were falling to the right. He had nausea, but was not unconscious. This attack did not recur but the sensation of tumbling to the right remained.

Examination on October 11th, 1922, revealed the following facts:

The right ear drum was partly calcified and turbid, the left one showed no special remnants of the former otitis media. A septum deviation to the right was present. The reactions of WASSERMANN and SACHS GEORGI were negative.

*Acoustic function:* The whispering voice was heard at 2 M at the right side, at  $\frac{1}{2}$  M at the left side. The tuning fork test proved a bilateral labyrinthine deafness to be present. The test of Rinne was positive, the bone conduct was shortened.

<sup>1)</sup> Examination on nystagmus was, for all patients, performed always when looking straight forward, and in the last years, also with the spectacles of Frenzel.

*Vestibular nystagmus:* No spontaneous nystagmus was present, except a slight fixation-nystagmus after looking to the right. No spontaneous past pointing in the different planes. The test of ROMBERG was negative, walking with closed eyes was possible without disturbances. It was impossible to elicit a fistula-symptom.

*Rotatory nystagmus.* The horizontal nystagmus, both after turning to the right and to the left was equal and of normal duration, (28 respectively 26 sec.). The reactive past pointing was bilaterally normal, but weak.

The rotatory nystagmus was after turning to the right and the left also equal and of normal duration (14, resp. 16 sec.).

The vertical nystagmus, however, showed disturbances; after turning to the right, with the patient in the right lateral position, a distinct vertical nystagmus downward, was produced and proved to be of normal duration; the after-nystagmus however did not appear after turning to the left; on repeated examination only some nystagmus movements could be noted.

These last observations are the more remarkable because other symptoms pointed to an allergy for the vertical types of nystagmus. When the patient was changed from the sitting into the dorsal position, a strong vertical nystagmus upward and rotatory to the left developed after a short pause of  $\pm 10$  sec. This attack was only of short duration although patient was very dizzy. Turning the patient from the dorsal to the sitting position exactly the reverse took place: also after a short pause of  $\pm 10$  sec. a distinct nystagmus combined with dizziness was produced: now however its direction was downward, while a rotatory component to the right was present<sup>1</sup>).

The caloric reaction was erroneously only examined with cold water. The bilaterally obtained reactions were typical: a nystagmus was produced with its direction to the side which was not douched.

Compensatory eye-positions: these amounted in right lateral position to 7°, in left lateral position to 3°.

Position nystagmus:

- I. Patient completely in right lateral position: no nystagmus.
- II. Trunk in dorsal position, head in right lateral position: no nystagmus.
- III. Patient completely in left lateral position: no nystagmus.
- IV. Trunk in dorsal position, head in left lateral position: horizontal rotatory nystagmus to the left.

Although the former otitis made a posterior arachnoiditis probable, this diagnosis could not be made with certainty especially because extensive neurological examination and further investigation were impossible, as the patient did not return.

In the following two cases concerning a position nystagmus elicited by "Neck-reflexes", the cause of this nystagmus remained completely obscure.

The first of these two cases is a patient in whom the above mentioned syndrome was first seen.

Mrs. G., 54 years.

For years patient suffered of right-sided tinnitus. About one year ago she had an attack of dizziness; then the whole room seemed to be turning round. This attack lasted for 5 minutes, no nausea developed. Shortly after the attack recurred. Besides she had

<sup>1</sup>) These attacks of nystagmus are in line with those described by BÁRÁNY: "durch rasche Kopfbewegungen ausgelösten Nystagmusanfälle (W. med. Wochenschr. nr 4, 1910). As far as my opinion goes the explanation of these "Nystagmusanfälle" meets great difficulties. However, it is here not the place to enter into such conjectures.

noted, when lying in bed and turning the head to the left, she became dizzy. When turning the head to the right side, no dizziness developed.

Examination on August 19th, 1922:

*Internal examination* (Dr. WOLVIUS): No disturbances except a slight dilatation of the heart to the left and a systolic râle at the top.

*Acoustic and rhinologic function*: no disturbances. The whispering voice was heard at normal distances.

*Vestibular function*: no spontaneous nystagmus or past pointing. The horizontal nystagmus, after turning to the right and to the left, was normal and of equal duration (20 and 19 sec.). Reactive past pointing on both sides typical.

The caloric examination of both labyrinths proved that typical reactions were present.

Position nystagmus:

- I. Patient with head and trunk in dorsal position: no nystagmus.
- II. Trunk in dorsal position, head in right lateral position: no nystagmus.
- III. Trunk in dorsal position, head in left lateral position: strong rotatory nystagmus to the right, somewhat diagonal, combined with vertigo. These symptoms remained as long as head and trunk were kept in this position.
- IV. Patient completely in left lateral position: no nystagmus.
- V. Head in left lateral position and trunk turned to dorsal position: Immediately a strong vertigo and a rotatory nystagmus to the right were produced.

Conformable symptoms appeared when, in sitting position, the position of the head in relation to the trunk, was changed, by turning it to the right or to the left side. No nystagmus, however, developed after pressure on the vessels of the neck.

Administration of 0.5 gram of urotropine daily, during 2½ months improved her condition. Hereafter neither dizziness nor nystagmus could no more be elicited by turning the head. The condition remained well after discontinuance of this treatment.

**Mrs. F., 57 years.**

11 Years previously patient developed an influenza, attacks of vertigo and vomiting. Sometimes the room went "up and down", on occasion she had the feeling to fall with her bed in depths. The hearing function of the left ear was bad, tinnitus of this ear was always present.

The above mentioned attacks of "Ménière" appeared occasionally during 6 years, hereafter she was very well until 3 months prior to examination when the attacks recurred. Three weeks ago they became much more pronounced and frequent so that she became bed-ridden. The hearing function of the right ear had also become worse.

Examination on October 28th 1938 (with her physician) proved that no internal disturbances were present; the blood pressure was completely normal.

Superficial examination of the patient lying in dorsal position, revealed no nystagmus after looking in all directions. If, however, the head or the patient was turned to the right lateral position, a horizontal nystagmus to the right was produced.

Patient was advised to come to the clinic for an extensive examination, but refused. However, in June 1939, a psychologic shock caused a tinnitus of the right ear, lasting 14 days; it was accompanied by a serious attack of vertigo. Shortly after this a second attack followed, the tinnitus remained, slight attacks of dizziness appeared and patient felt as though she were falling to the right. She now asked to be admitted to the clinic.

*Neurological examination* (Dr. DE JONG): Extensive examination revealed no disturbances. The fundi were normal. The cerebrospinal fluid was clear and did not contain abnormal substances. The reactions of WASSERMANN and SACHS GEORGI in blood and cerebrospinal fluid were negative.

*Acoustic function*: Whispering voice was only heard at 0.3 M at both sides, this was caused by a labyrinthine deafness.

*Vestibular function*: No spontaneous nystagmus or past pointing. The caloric reaction of both labyrinths by douching the ears with cold and warm water was present but weak.



A marked "Nystagmusbereitschaft" was found for a nystagmus with its quick component to the left (the right labyrinth was especially reactive for cold, the left one especially for warm water).

Position nystagmus:

- I. Trunk and head in dorsal position: no nystagmus.
- II. Patient completely in right lateral position: no nystagmus.
- III. Patient completely in left lateral position: no nystagmus.
- IV. Trunk in dorsal position, head in right lateral position: horizontal nystagmus to the left.
- V. Trunk in dorsal position, head in left lateral position: horizontal nystagmus to the right.

It was remarkable that the first examination revealed a nystagmus of the labyrinthine type (nystagmus with the patient completely in lateral position). A second clinical examination, however, only revealed a nystagmus when, with the head in lateral position, the position of the head was changed in relation to the trunk.

The explanation of these phenomena could be as follows: in the first period of the disease a lateral position was sufficient to cause a nystagmus; later (second examination) the position nystagmus could only be produced if two stimulations were present, i.e. a labyrinthine stimulation by the lateral position of the head and a second one, caused by the position of the head in relation to the trunk.

However, we have pointed out already, in relation to a similar conception of BORRIES (see page 154) that a more simple way to establish the fact, if the labyrinthine factor is also necessary to produce the nystagmus, is placing the patient in the sitting position and to see if in the different positions obtained by turning the head, a nystagmus develops.

By this way of examination the labyrinthine factor is namely eliminated. We did not perform this examination. As the patient left for a foreign country, the further course is also unknown.

### *Summary.*

Six cases of a particular form of position nystagmus are described. This form is characterized by the development of the nystagmus after changing the position of the head in relation to the trunk.

It was proved that this type of nystagmus can be elicited in cases of posterior arachnoiditis. The stimulation, necessary for the development of the nystagmus in these cases of posterior arachnoiditis, is probably due to disturbances in the liquor circulation, which are caused by the turning of the neck.

Manifestation of this form of position nystagmus in suspected cases of posterior arachnoiditis might be of some diagnostic value.

To elicit such a position nystagmus neither labyrinthine factors nor disturbances of the circulation in the jugular vein or in the carotid artery caused by turning of the neck, are necessary. It is remarkable that in two of the six described cases a "Nystagmusbereitschaft" was present, while in the same cases a, — be it temporary — labyrinthine form of position nystagmus could be demonstrated.

**Mathematics.** — *The analogy between the statistics of numbers and statistical mechanics.* By L. S. ORNSTEIN and J. M. W. MILATZ.

(Communicated at the meeting of January 25, 1941.)

### § 1. *Introduction.*

In classical physics statistical considerations ought to be introduced, if complicated systems are studied.

The equations of motion describing the change of the systems as a function of time are known in principle, but statistics must be introduced as it is impossible to overlook the conclusions drawn from these equations neither mathematically nor physically; so it is for example impossible to survey in detail the initial state of a system composed of molecules or to follow its change with the time.

Now the same is true for the regularities present in the order of the figures of a given irrational number, they must exist but are seldom known explicite. This has been the motive for the introduction of statistical considerations for the figures of those numbers. Considerations of this point already have been given by H. POINCARÉ (*Science et Méthode*) and rather fully by E. BOREL <sup>1)</sup>.

In physics two methods have been used the older one the kinetical theory of gases treats defined problems; in the ingenious hand of BOLTZMANN it has been developed to great generality, the second method is the more axiomatic treatment of J. W. GIBBS, who never discusses single systems but takes ensembles and investigates the properties of the means in those ensembles and of the systems most frequently occurring in the ensembles. The analogy of the physical probability and the thermodynamical quantities is put very sharp by the work of GIBBS. We will try to show that an analoqueous treatment is possible for the problems of the statistical theory of numbers. In this way we will get in an another way some of the results of BOREL — this was not our principle aim — it is to demonstrate the bearing of GIBBS' method on this type of problems of the theory of numbers. Others have already shown that the use of ensembles enables to formulate a theory of probability without the help of games or regions of equal probability, we have tried to use these method as consequent as possible. To the physicist it will be appearant, that our considerations have some relation with the image of steps given by VON

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<sup>1)</sup> E. BOREL. *Valeur pratique et philosophie des probabilités applications à l'arithmétique et à la théorie des fonctions.*

*Traité du Calcul. des Probabilités et de ses applications.* Tome II fasc. I and Tome IV fasc. III. Paris Gauthier-Villars.





So we have  $\overline{n^2} - \bar{n}^2 = \overline{(n - \bar{n})^2} = \frac{N}{4}$  and thus

$$s = \frac{\overline{(n - \bar{n})^2}}{\bar{n}^2} = \frac{1}{N}. \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (4)$$

The quantity  $s$  is a measure for the scattering of the values of  $n$  relatively to the mean value  $\bar{n}$ .

The larger we take  $N$  the smaller the quantity  $s$  is. If  $N$  goes to infinite  $s$  gets the value zero. Now taking into account that  $\bar{n} = \frac{N}{2}$  means that the zero and the one are equally occurring in the mean, we can interpret our result in such a way, that the preponderant majority of the ensemble under consideration consist of numbers in which the one and the zero have equal occurrence. BOREL has called those numbers "normal" numbers. We thus obtain the same result as BOREL: *the preponderant majority of the numbers in the continuum between zero and one is normal*. Thus for the case that  $N$  is infinite choosing a number ad random there is an overwhelming probability that this number consist of an equal number of 0 and 1. As in this way as a rule an irrational number is obtained we come to the conclusion that irrational numbers are normal as a rule. Drawing this conclusion we have assumed — as happens as a rule in the applications — testings the laws of probability with reality, that choosing ad random a system is obtained showing, the properties of the most frequently occurring or the mean system.

We can formulate our conclusion still in another way.

We consider the fractions of the  $2^N$  systems for which the number of zero's is:  $n$  that of  $1 : N - n$ , this fraction can be defined as the probability of the  $n : N - n$  systems in the ensemble  $W(n)$ . As for large values of  $N$  the value of  $s$  is small, it has sense to discuss the question whether values of  $n$  differing markedly from  $\bar{n}$  occur still to some extend in the ensemble. We determine thus the probability for

$$n = \bar{n} + \vartheta$$

$\vartheta$  being small compared with  $\bar{n}$ . Now  $\bar{n} = \frac{N}{2}$  and  $N$  being large we can develop the faculties so that

$$W(n) = \frac{1}{2^N} \frac{N!}{(N-n)! n!}$$

transforms to

$$W(\bar{n} + \vartheta) = e^{-\frac{2\vartheta^2}{N}} = e^{-\frac{\vartheta^2}{\bar{n}}}. \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (5)$$

Putting  $\vartheta = \varrho \bar{n}$  we obtain

$$W(\bar{n} + \vartheta) = e^{-e^{\vartheta} \bar{n}} = W(\varrho). \quad . \quad . \quad . \quad . \quad . \quad (6)$$

The mean value  $\bar{n}$  is very large thus  $W(\varrho)$  shows only a value differing from zero in the immediate vicinity of  $\varrho = 0$ .

If  $N$  is infinite  $\bar{n}$  is also infinite, the frequency law  $W(\varrho)$  for the numbers between 0 and 1 is a  $\delta$ -function. The probability for  $\varrho$  to lay between  $\varrho$  and  $\varrho + d\varrho$  is represented by the formula

$$W(\varrho) d\varrho = \sqrt{\frac{\bar{n}}{\pi}} e^{-e^{\vartheta} \bar{n}} d\varrho. \quad . \quad . \quad . \quad . \quad . \quad (7)$$

The mean square of  $\varrho : \overline{\varrho^2} = \frac{1}{2\bar{n}}$  or  $\frac{1}{N}$ . This value which is obtained by approximation (development of the faculties) is in accordance with (4) which is exact; the cases neglected in our approximation dont play any part in the ensemble.

Formula (7) shows that the most frequently occurring number in the ensemble is normal; mean value and most frequent value are the same.

We can deduce the formula's obtained in another way giving further insight in the problem. We have defined  $Z(N, n)$  for an ensemble of fractions with  $N$  figures. We now add a figure and ask for  $Z(N + 1, n)$ . It is clear that we have:

$$Z(N + 1, n) = Z(N, n) + Z(N, n-1) \quad . \quad . \quad . \quad . \quad (8)$$

for the numbers are obtained putting 0 and 1 in all thinkable ways behind the numbers of the group  $Z(N, n)$ .

Introducing again the probability as defined above taking

$$W(N + 1, n) = \frac{1}{2^{N+1}} Z(N + 1, n)$$

we obtain from (8)

$$W(N + 1, n) = \frac{1}{2} W(N, n) + \frac{1}{2} W(N, n-1) \quad . \quad . \quad . \quad (9)$$

a known recurrent equation, which is generally found in introducing the probability  $\frac{1}{2}$  — for a zero or a one — which thus is not necessary in our way of proceeding.

Putting

$$W(N + 1, n) - W(N, n) = \frac{1}{2} W(N, n-1) - \frac{1}{2} W(N, n)$$

and taking  $N$  very large, we get

$$\frac{dW(N, n)}{dN} = \frac{1}{2} W(N, n-1) - \frac{1}{2} W(N, n)$$

or introducing  $\frac{N}{2} = \bar{n}$ ,

$$\frac{dW(\bar{n}, n)}{d\bar{n}} = W(\bar{n}, n-1) - W(\bar{n}, n). \quad (10)$$

The solution of this equation is the known POISSON formula:

$$W(\bar{n}, n) = \frac{(\bar{n})^n}{n!} e^{-\bar{n}}, \quad (11)$$

a formula giving the probability also for small values of  $n$ . The formula shows again that for the ensemble between 0 and 1 ( $N$  is infinite) deviations of normal numbers are extremely seldom.

Then taking  $\bar{n}$  very large and assuming small deviations  $n$  from  $\bar{n}$  only occur we get for the frequency law  $W(\bar{n}, n)$  a differential equation of the form:

$$\frac{\partial W}{\partial \bar{n}} = \frac{1}{2} \frac{\partial^2 W}{\partial n^2}$$

the known equation of diffusion having the GAUSSIAN law as its solution. It ought to be urged to the fact that our considerations are determined entirely by the construction of the ensemble and don't involve special hypotheses on probabilities.

### § 3. *The frequency law in the decimal system.*

In order to show the general character of our considerations we will work them out for the decimal system.

We again construct all numbers with  $N$  decimals situated between 0 and 1. In this way we obtain  $10^N$  numbers. We consider a number containing  $n_0$  times a zero,  $n_1$  times a one, ...,  $n_9$  times a nine, so that

$$\sum_0^9 n_\nu = N. \quad (12)$$

The function  $Z(.n_\nu.)$  giving the numbers of this type amounts to

$$Z(.n_\nu.) = N! \prod_0^9 \frac{1}{n_\nu!}. \quad (13)$$

Taking the sum of (13) for all values of  $n_\nu$  fulfilling (12), we obtain the total number  $10^N$ .

The mean value of the sum of the decimals for the numbers of the ensemble is

$$\frac{1}{10^N} \sum n_\nu \sum_0^9 n_\nu Z(.n_\nu.). \quad (14)$$



The value of (14) is  $4.5 N$ , the mean value of a decimal therefore is  $4.5^1$ ).

The mean square of the sum of the decimals can easily be deduced. We have

$$\overline{v^2} = \sum n_v (\sum v n_v)^2 Z(.n_v.) = \frac{N}{10} \sum_0^9 v^2 + \frac{N(N-1)}{10^2} \left( \sum_0^9 v \right)^2$$

or

$$\overline{(v-v')^2} = \frac{N}{10} \left\{ \sum_0^9 v^2 - \frac{1}{10} \left( \sum_0^9 v \right)^2 \right\}.$$

We thus get

$$\frac{\overline{(v-v')^2}}{v^2} = \frac{10}{N} \left( \frac{\sum_0^9 v^2}{\left( \sum_0^9 v \right)^2} - \frac{1}{10} \right) \dots \dots \dots (16)$$

From the formula (16) it is evident that our ensemble consist chiefly of normal numbers if we take  $N$  infinite. Taking  $N$  very large and assuming

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<sup>1)</sup> This result can be deduced in the following way. Taking as a starting point the identity

$$\begin{aligned} (a_0 + a_1 + \dots a_9)^N &= \sum n_v \frac{N!}{n_0! \dots n_9!} a_0^{n_0} \dots a_9^{n_9} = \\ &= \sum n_v Z(.n_v.) a_0^{n_0} \dots a_9^{n_9} \end{aligned}$$

and differentiating with respect to  $a_v$  this formula we get

$$N(a_0 + \dots a_9)^{N-1} = \sum n_v Z(.n_v.) n_v a_0^{n_0} a_v^{n_v-1} \dots a_9^{n_9} \dots \dots (15)$$

Taking  $a_0 = a_1 = \dots a_9 = 1$  we have

$$\frac{N}{10} = \frac{1}{10^N} \sum Z(.n_v.) n_v = \bar{n}_v.$$

The mean decimal therefore is

$$\sum_0^9 v \bar{n}_v = \frac{N}{10} \sum_0^9 v = 4.5 N.$$

In the same way  $\overline{n_v^2}$  and  $\overline{n_v n_{v'}}$  can be determined if we multiply (15) by  $a_v$  and differentiate respectively to  $a_v$  and  $a_{v'}$ . We then find

$$\overline{n_v^2} = \frac{N}{10} + \frac{N(N-1)}{10^2}$$

$$\overline{n_v n_{v'}} = \frac{N(N-1)}{10^2}.$$

that the deviations of the  $n_v$ 's from their mean values are small we obtain as the frequency law for the  $q_v$   $q_v$ 's,  $= \frac{n_v - \bar{n}_v}{\bar{n}_v}$ ,

$$W(.q_v.) \propto e^{-5N \sum_0^9 q_v^2}$$

where  $\sum_0^9 q_v = 0$ . This law shows again how strongly the normal numbers are present if  $N$  tends to infinity. Considerations for each decimal  $v$  can be developed in the same way.

#### § 4. Discussion of the occurrence of the figures 0 and 1 in the fraction of the binary ensemble.

The  $2^N$  numbers of the ensemble are arranged along their magnitude and divided in groups in the following way. The first number has  $N$  figures zero, the second  $N-1$  times a zero with one 1 at the end, the next  $N-2$  times a zero followed by 10 or 11 the third group  $N-3$  times a zero followed by either 100, 101, 110 or 111, in general  $N-p$  times a zero then 1 followed by all possible combinations of 0 and 1 (beginning with 0,  $N-p$  times zero, 1  $p-1$  times zero and ending with 0  $N-p$  times zero, 1  $p-1$  times one), the group contains  $2^{p-1}$  numbers totally. So  $p=N$  so we get 0.1 (decimal system 0.5). The further groups can be formed from those we already have mentioned by interchange of 0 and 1.

We can graphically represent the numbers dividing a line so that the steps between the numbers agree with a jump of  $2^{-N}$  existing between consecutive numbers. Taking a line element large compared with these steps we find a certain number of numbers pro unity of length. This is still the case if  $N$  gets larger and larger, we can however still speak of  $q(x)$ , the density of the distribution of the numbers, also for the case of the limit that  $N$  is infinite.

The ensemble under consideration has a constant value for  $q(x)$ ; we will see in the following § that this is not the only possible case.

We now investigate the statistic of the figure zero in the  $p^{\text{th}}$  group. This group contains first by  $N-p$  times zero and further a combination of zero and one's. We find a group of numbers with  $k+1$  times one consisting of

$$\frac{(p-1)!}{(p-1-k)! k!}$$

numbers. The total number of zero's then is  $(N-p) + p - (1+k) = N-k-1$ . The mean value of the number of zero's of the group consist of two parts i.e.  $(N-1) 2^{p-1}$  minus  $k$  or  $(p-1) 2^{p-2}$ . As the group contains  $2^{p-1}$  numbers the mean number of zero's pro member of the group amounts to  $N - \frac{p+1}{2}$ .

In order to get an idea of the distribution of numbers we can plot the first point of the group as an abscis against the mean number of zero's as an ordinate. If a group of  $2^{-N}$  corresponds with a length  $l$  we find  $2^p-1$  times  $l$  as the abscis of the  $p^{\text{th}}$  group, the ordinate is  $x = (2^p-1)l$ . For  $p=N$  we get 0.1 that is the centre of the interval, thus  $2^N-1$  preces  $l$ . With  $2^p-2$  corresponds a number  $N - \frac{p+1}{2} = y$  of zero's. The relation between  $x$  and  $y$  thus can be indicated.

If we take  $N$  large and  $p$  not small we have as a proportion  $\nu$  of the abscisses given by  $\nu = 2^{p-N}$  or  $p - N = \frac{\lg \nu}{\lg 2}$ . The number of zero's for a given ratio  $\nu$  thus is  $\frac{N}{2} - \frac{1}{2} \frac{\log \nu}{\log 2}$ . This formula shows that for the preponderate part of the interval the proportion is practically  $\frac{N}{2}$ . If we ask for which point the number of zero's is  $0.75 N$  we find that  $\nu = 2^{-0.25 N}$  that is extremely near to the point zero.

### § 5. Ensembles (more general discussion).

We find in statistical mechanics that two canonical ensembles can be combined to another with the same modulus, if we combine the systems of both ensembles of equal modulus. We will investigate whether the ensembles of numbers show an analoqueous property. We take two ensembles of  $N$  decimals and make the combination considering all the possible products of the members of both ensembles. Let  $x$  denote the numbers in the one,  $y$  those in the other ensemble, the products thus are  $xy = a$ . If  $\varrho(x)$  is the density of systems in the first and  $\varrho(y)$  that in the other ensemble  $xy = a$  will show a value between  $a$  and  $a + da$  for

$$\varrho(x) \varrho(y) dx dy \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (17)$$

systems; the form (17) ought to be integrated for the region where  $a$  has a value between  $a$  and  $a + da$  in order to get the systems in the new ensemble showing a value of  $a$  between the limits indicated.

We can represent the whole in a square  $xy$  with the sides 1 in a region between the hyperbola  $xy = a$  and  $xy = a + da$  is constant, this region amounts to  $-\lg a da$ , taking  $\varrho(x) = \varrho(y) = \text{constant} = \varrho$  we have for the number of systems in the new ensemble which are situated between  $a$  and  $a + da$ .

$$-\varrho^2 \lg a da \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (18)$$

The total number in the ensemble is  $\varrho^2$ , which follows integrating (18) with respect to  $a$  from 0 to 1. The ensemble originated is an example of a more complicated type; however still the overwhelming majority of its numbers is normal.



As a second example we take the case that the numbers of two ensembles are added. We have now in the new ensemble  $\alpha = x + y$  the region where

$$\varrho^2 dx dy$$

must be taken together in order to have the systems between  $\alpha$  and  $\alpha + d\alpha$  is a strip between two lines inclined under 45 degrees in the square mentioned above. The magnitude of the strip is  $\alpha d\alpha$  for the left side of the diagonal and  $(\alpha' - 1)d\alpha'$  for the right side. We now remark that two numbers with the same figures behind the comma, differing however one before it takes equal thus of  $\alpha' - 1 = \alpha$ . Taking those numbers as identical, it is clear that the whole square is homogeneously filled with the systems of the ensemble. The ensemble generated from the two ensembles by taking the sum therefore is of the same type as the original ensembles. Now the same is true if we do no longer take  $\varrho$  independent of  $x$  or  $y$  but assume  $\varrho(x) \propto e^{-\frac{x}{\theta}}$  and  $\varrho(y) \propto e^{-\frac{y}{\theta}}$ . The number of systems is than

$$e^{-\frac{x+y}{\theta}} dx dy$$

integrated along the trips described, where  $x + y$  is constant. If we assume that  $x + y$  and  $x + y + 1$  ought to be identified the ensembles under consideration are transformed to an one ensemble of the same type by addition of their numbers.

It is in reality possible to show that it has all sense to introduce ensembles of the type given. We take again the  $2^N$  numbers of  $N$  figures and imagine a distribution with density  $\varrho(x)$  on the interval  $0 \dots 1$ . The density  $\varrho$  is the statistical weight of the numbers. (Implicately we always can go to the limit  $N$  infinite.) We have:

$$\int_0^1 \varrho(x) dx = C. \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (18)$$

Further we assume that the ensemble is characterised by the mean value of the number  $x$  such that

$$\int_0^1 x \varrho(x) dx = CE \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (19)$$

$E$  being the mean value (energy of the ensemble).

Let we consider the number of distributions  $\varrho(x)dx$  which are possible with the  $C$  systems, situated on the length  $0 - 1$ . This number is

$$Z = \frac{C!}{\Pi (\varrho(x) dx)!}.$$

For which distribution  $Z$  is maximum? In order to find the distribution we take the logarithm and develop the faculties. Than

$$H = \int_0^1 \varrho(x) \log(\varrho(x)) dx. \quad . \quad . \quad . \quad . \quad . \quad (20)$$

ought to be minimum, this is the  $H$  theorem in the theory of numbers.

Applying calculus of variation and taking into account (18) and (19) we find

$$0 = \int_0^1 \delta \varrho \left( 1 + \log \varrho(a) + v + \frac{x}{\theta} \right)$$

or

$$\varrho(x) = A e^{-\frac{x}{\theta}}$$

The constants  $A$  and  $\theta$  can be determined with the help of (18) and (20). Comparing all ensembles of given  $E$  and  $C$  the canonical ensemble is the most probable. For  $\theta = 0$  and  $E = \frac{1}{2}$  we come back to the original ensemble.

*Utrecht*, Dec. 1940, Jan. 1941.

**Mathematics.** — *Elemente der intuitionistischen Funktionentheorie*<sup>1)</sup>.  
(Erste Mitteilung.) *Die CAUCHYschen Integralsätze und die TAYLORsche Reihe.* By M. J. BELINFANTE. (Communicated by Prof. L. E. J. BROUWER.)

(Communicated at the meeting of January 25, 1941.)

§. 1. Im folgenden ist mit  $G$  eine beschränkte Punktmenge in der komplexen Ebene gemeint. Ist jedem Punkt  $z$  aus  $G$  eine komplexe Zahl  $F(z)$  zugeordnet und lässt sich dabei zu jedem positiven  $\varepsilon$  eine solche positive Zahl  $d = d(\varepsilon)$  bestimmen, dass:

$$\left| \frac{F(z_3) - F(z_1)}{z_3 - z_1} - \frac{F(z_2) - F(z_1)}{z_2 - z_1} \right| < \varepsilon \quad . \quad . \quad . \quad . \quad . \quad (1)$$

ist für irgend drei Punkte  $z_1$ ,  $z_2$  und  $z_3$  aus  $G$  die den Bedingungen:

$$0 < |z_1 - z_2| < d \text{ und } 0 < |z_1 - z_3| < d$$

genügen, so heisst  $F(z)$  eine in  $G$  reguläre Funktion.

Ist  $G$  ein reelles Intervall ( $a \leq t \leq b$ ), so bilden die zugeordneten Funktionswerte  $F(t)$  in der komplexen Ebene einen Weg  $F(t)$ , wobei wir sagen dass der Punkt  $F(t_1)$  zwischen  $F(t_2)$  und  $F(t_3)$  liegt, falls  $t_1$  zwischen  $t_2$  und  $t_3$  liegt. Gilt für eine endliche Anzahl Wege  $F_i(t)$  mit den Intervallen  $a_i \leq t \leq b_i$  die Beziehung  $F_{i+1}(a_{i+1}) = F_i(b_i)$ , so bilden sie zusammen eine Punktmenge, die ebenfalls ein Weg genannt wird. In dem letzten Fall darf ohne wesentliche Beschränkung  $a_{i+1} = b_i$  vorausgesetzt werden. Die Punkte eines Weges brauchen nicht von einander entfernt zu sein.

In den folgenden Sätzen ist  $G$  ein Gebiet.

**Satz 1.** *Eine in  $G$  reguläre Funktion  $F(z)$  ist dort beschränkt d.h. es lässt sich eine positive Zahl  $M$  so bestimmen, dass die Ungleichung  $|F(z)| < M$  für jeden Punkt aus  $G$  erfüllt ist.*

*Beweis.* Man wähle in  $G$  eine endliche Anzahl Punkte  $Z_i$  in solcher Weise, dass jeder Punkt aus  $G$  von wenigstens einem dieser Punkte eine Entfernung  $< d = d(1)$  hat und ordne jedem  $Z_i$  einen derartigen Punkt  $\zeta_i$  aus  $G$  zu, dass  $0 < |Z_i - \zeta_i| < d$  ist. Sei  $\mu > 1$  eine solche positive Zahl, dass die Ungleichung:

$$\left| \frac{F(Z_i) - F(\zeta_i)}{Z_i - \zeta_i} \right| < \mu - 1$$

<sup>1)</sup> Diese Reihe von Aufsätzen soll eine vollständige Herleitung meiner in diesen Proceedings Bd. 34 (1931), S. 1395—1397 mitgeteilten Resultate bringen.



für jedes  $i$  erfüllt ist. Bestimmt man nun noch ein solches  $M > \mu d$ , dass die Ungleichung:  $|F(Z_i)| < M - \mu d$  für jedes  $i$  erfüllt ist, so ist  $F(z)$  in  $G$  dem absoluten Betrage nach kleiner als  $M$ .

**Satz 2.** Eine in  $G$  reguläre Funktion  $F(z)$  ist dort gleichmässig stetig, d.h. es lässt sich zu jedem  $\varepsilon > 0$  ein solches  $\eta = \eta(\varepsilon) > 0$  bestimmen, dass die Ungleichung  $|F(z_1) - F(z_2)| < \varepsilon$  erfüllt ist, sobald die Punkte  $z_1$  und  $z_2$  aus  $G$  der Bedingung  $|z_1 - z_2| < \eta$  genügen.

**Beweis.** Wählt man im vorigen Beweis bei vorgegebenem  $\varepsilon > 0$  eine positive Zahl  $\eta$ , die kleiner ist als die beiden positiven Zahlen  $d$  und  $\frac{\varepsilon}{\mu + 1}$ , so gilt  $|F(z_1) - F(z_2)| < \varepsilon$ , sobald  $|z_1 - z_2| < \eta$  ist.

**Satz 3.** Eine in  $G$  reguläre Funktion  $F(z)$  hat dort eine Ableitung  $F'(z)$ , d.h. in jedem Punkt von  $G$  lässt sich eine Zahl  $F'(z)$  bestimmen mit der Eigenschaft, dass bei jedem vorgelegtem  $\varepsilon > 0$  eine positive Zahl  $\delta = \delta(\varepsilon)$  existiert, sodass die Ungleichung:

$$\left| \frac{F(z') - F(z'')}{z' - z''} - F'(z'') \right| < \varepsilon \quad . \quad . \quad . \quad . \quad . \quad (2)$$

erfüllt ist für irgend drei Punkte  $z', z''$  und  $z'''$  aus  $G$ , welche den Bedingungen  $0 < |z' - z''| < \delta$  und  $0 < |z' - z'''| < \delta$  genügen.

Umgekehrt ist eine Funktion in  $G$  regulär, sobald sie dort eine Ableitung besitzt.

**Beweis.** Sei  $\varepsilon_1 > \varepsilon_2 > \dots$  eine monotone Nullfolge. Wir bestimmen zuerst in einem vorgelegten Punkt  $Z$  von  $G$  die Zahl  $F'(Z)$ . Wir bestimmen die Folge  $z_1, z_2, z_3, \dots$  derart, dass: a.  $|Z - z_n| < d(\varepsilon_n)$  ist, b.  $|Z - z_i| > |Z - z_{i+1}|$  ist und c.  $Z - z_n$  gegen Null strebt. Setzen wir nun für  $z \neq Z$ :  $g(z) = \frac{F(Z) - F(z)}{Z - z}$ , so ist  $|g(z_n) - g(z_{n+p})| < \varepsilon_n$ .

Mithin existiert  $F'(Z) = \lim_{n \rightarrow \infty} g(z_n)$ . Gilt weiter  $0 < |z' - z''| < d(\varepsilon_n)$  und  $0 < |z' - z'''| < d(\varepsilon_n)$ , so folgert man leicht, dass (2) mit  $3\varepsilon_n$  statt  $\varepsilon$  erfüllt ist. Bestimmt man also bei vorgelegtem  $\varepsilon > 0$  die ganze Zahl  $n$  derart, dass  $3\varepsilon_n < \varepsilon$  ist, so genügt es für  $\delta(\varepsilon)$  die Zahl  $d(\varepsilon_n)$  zu nehmen.

Für den Beweis, dass umgekehrt eine Funktion in  $G$  regulär ist, sobald sie dort eine Ableitung hat, braucht man nur bei vorgelegtem  $\varepsilon > 0$  für die gesuchte Zahl  $d(\varepsilon)$  die Zahl  $\delta(\frac{1}{2}\varepsilon)$  zu wählen.

**Satz 4.** Die Ableitung einer in  $G$  regulären Funktion ist dort gleichmässig stetig.

**Beweis.** Folgt unmittelbar aus (2).

**Satz 5.** Eine in  $G$  gleichmässig stetige Funktion  $F(z)$  ist dort beschränkt.

**Beweis.** Sei  $|F(z') - F(z'')| < 1$  für  $|z' - z''| < \eta$ . Bestimmt man

eine endliche Anzahl Punkte  $Z_i$  in solcher Weise, dass jeder Punkt aus  $G$  von wenigstens einem dieser Punkte  $Z_i$  eine Entfernung  $< \eta$  hat und bestimmt man die positive Zahl  $M > 1$  derart, dass die Ungleichung  $|F(Z_i)| < M - 1$  für jedes  $i$  erfüllt ist, so gilt  $|F(z)| < M$  für jeden Punkt aus  $G$ .

§ 2. Wählt man zu einem Weg  $z(t)$  ( $a \leq t \leq b$ ) die reellen Werte  $t_1, t_2, \dots, t_n$  derart, dass  $a = t_0 < t_1 < t_2 < \dots < t_n < t_{n+1} = b$  ist, so heisst der Ausdruck  $\sum_0^n |z(t_{i+1}) - z(t_i)|$  die Länge des der  $t$ -Zerlegung  $(t_i)$  zugeordneten Sehnepolygons von  $z(t)$  zwischen  $a$  und  $b$ . Eine Fundamentalreihe  $t$ -Zerlegungen  $\{t_i^p\}$  heisst einfach, falls:

a. jedes  $t_i^p$  einer folgenden Zerlegung entweder einem  $t_j^{p-1}$  der vorigen Zerlegung gleich ist oder zwischen zwei Werte  $t_j^{p-1}$  und  $t_{j+1}^{p-1}$  dieser Zerlegung liegt, und jedes  $t_k^{p-1}$  einem  $t_l^p$  gleich ist,

b. zu jeder Zerlegung  $(t_i^p)$  eine solche positive Zahl  $l_p$ , die Breite der Zerlegung, bestimmt werden kann, dass  $|t_{i+1}^p - t_i^p| < l_p$  und  $\lim l_p = 0$  ist.

**Satz. 6.** Die Längen der den Zerlegungen einer einfachen Fundamentalreihe zugeordneten Sehnepolygone eines Weges  $z(t)$  konvergieren zu einem Limes, der unabhängig ist von der gewählten einfachen Fundamentalreihe. Es heisst dieser Limes  $L$  die Länge des Weges  $z(t)$  zwischen  $a$  und  $b$ , bzw. zwischen  $z(a)$  und  $z(b)$ .

*Beweis.* Wählt man bei vorgegebenem  $\varepsilon > 0$  ein solches positives  $d$ , dass die Ungleichung:

$$\left| \frac{z(t''') - z(t'')}{t''' - t''} - z'(t') \right| < \frac{\varepsilon}{2(b-a)}$$

erfüllt ist, falls  $t'$ ,  $t''$  und  $t'''$  den Bedingungen  $0 < |t''' - t''| < d$  und  $0 < |t' - t''| < d$  genügen, so sind zwei Zerlegungen einer beliebigen einfachen Fundamentalreihe, deren Breiten kleiner als  $d$  sind, Sehnepolygone zugeordnet, deren Längen sich um weniger als  $\varepsilon$  unterscheiden.

**Satz 7.** Es sei  $L = z(t)$  ( $a \leq t \leq b$ ) ein Weg,  $\{t_i^n\}$  eine einfache Fundamentalreihe  $t$ -Zerlegungen und  $f(z)$  eine längs  $L$  gleichmässig stetige Funktion. Alsdann konvergiert der Ausdruck

$$S_n = \sum f(\xi) (z_{i+1}^n - z_i^n), \text{ wo } z_i^n = z(t_i^n), \xi = z(\tau)$$

und  $\tau$  eine beliebige, der Ungleichung  $t_i^n \leq \tau \leq t_{i+1}^n$  genügende Zahl ist, zu einem Limes, der von der gewählten einfachen Fundamentalreihe unabhängig ist und der das längs  $L$  genommene Integral

$$I(L) = \int_{z(a)}^{z(b)} f(z) dz$$

heisst.

*Beweis.* Sei  $\lambda$  die Länge von  $L$  und  $l > \lambda$ . Bestimmt man zu einem gegebenem  $\varepsilon > 0$  ein solches  $\eta > 0$ , dass die Ungleichung  $|f(z'') - f(z')| < \frac{\varepsilon}{l}$  für  $|z'' - z'| < \eta$  erfüllt ist und wählt man  $d > 0$  derart, dass  $|z(t'') - z(t')| < \eta$  ist, sobald  $|t'' - t'| < d$  genommen wird, so sind die Werte von  $S_n$  für zwei Zerlegungen einer Fundamentalreihe, deren Breiten  $< d$  sind, um weniger als  $\varepsilon$  verschieden.

**Satz 8.** Für das in Satz 7 definierte Integral gelten folgende Beziehungen:

$$a. \int_{z(a)}^{Lz(b)} f(z) dz = - \int_{z(b)}^{Lz(a)} f(z) dz \quad (\text{Definition der linken Seite für } a > b)$$

$$b. \int_{z(a)}^{Lz(b)} f(z) dz + \int_{z(b)}^{Lz(c)} f(z) dz = \int_{z(a)}^{Lz(c)} f(z) dz$$

$$c. \int [c_1 f_1(z) + c_2 f_2(z)] dz = c_1 \int f_1(z) dz + c_2 \int f_2(z) dz$$

$$d. \left| \int_L f(z) dz \right| < Ml, \text{ falls } l \text{ die Länge von } L \text{ und } f(z) \text{ längs } L \text{ dem absoluten Betrage nach kleiner als } M \text{ ist.}$$

$$e. \int_{Z_1}^{Z_2} dz = Z_2 - Z_1$$

$$f. \int_{Z_1}^{Z_2} z dz = \frac{1}{2} (Z_2^2 - Z_1^2).$$

*Beweis von f.* Es ist

$$\int_{Z_1}^{Z_2} z dz = \lim \sum \frac{z_{i+1} + z_i}{2} (z_{i+1} - z_i) = \frac{1}{2} (Z_2^2 - Z_1^2).$$

§ 3. Ein Gebiet  $G$  mit der Eigenschaft, dass die inneren Punkte eines Vielecks  $V$  in  $G$  liegen, falls der Rand von  $V$  in  $G$  liegt, heisst *einfach-zusammenhängend*.

*Hauptsatz.* Es sei  $f(z)$  regulär in einem einfach-zusammenhängenden Gebiete  $G$  und es sei  $L$  ein in  $G$  liegender, geschlossener Weg, dessen

Punkte von jedem Randpunkt von  $G$  um mehr als eine feste positive Zahl  $a$  entfernt sind. Alsdann gilt:

$$I(L) = \int_L^L f(z) dz = 0.$$

*Bemerkung.* Nach dem BROUWERSchen Satz der finiten Spezies<sup>2)</sup> ist die Bedingung für den in  $G$  liegenden, geschlossenen Weg  $L$  erfüllt, falls jeder Punkt von  $L$  von jedem Randpunkt von  $G$  eine positive Entfernung hat.

*Beweis. Erster Fall.*  $L = L(z_1 z_2 z_3)$  besteht aus den Strecken  $z_1 z_2$ ,  $z_2 z_3$  und  $z_3 z_1$ , welche drei Punkte  $z_1$ ,  $z_2$  und  $z_3$  verbinden. Es brauchen die Punkte  $z_1 z_2 z_3$  nicht von einander entfernt zu sein oder sogar ein Dreieck zu bilden; mit der Strecke, welche  $z_1 z_2$  verbindet, ist der Weg  $z(t) = tz_2 + (1-t)z_1$ ;  $0 \leq t \leq 1$  gemeint. Setzt man für  $p = 1, 2, 3$ :

$$z_{p+3} = z_p ; z_{p,a} = z_p ; z_{p,b} = \frac{1}{2}(z_{p+1} + z_{p+2}),$$

so ist:

$$I(z_1 z_2 z_3) = I(z_{1,b} z_{2,b} z_{3,b}) + \sum_{i=1}^3 I(z_{i+2,a} z_{i+1,b} z_{i,b}).$$

Jedes Integral auf der rechten Seite lässt sich wieder als eine Summe von vier derartigen Integralen schreiben, usw. Auf der  $n$ -ten Stufe einer solchen Integralzerlegung ist das Integral  $I(z_1 z_2 z_3)$  dargestellt als die Summe von  $4^n$  Integralen  $I(\zeta_1 \zeta_2 \zeta_3)$  und es hat jeder Integrationsweg die Länge  $\frac{l}{2^n}$ , wo  $l$  die Länge von  $L(z_1 z_2 z_3)$  ist. Zu einem vorgegebenem  $\varepsilon > 0$  lässt sich nun  $d > 0$  derart bestimmen, dass:

$$|f(Z_1) - f(Z_2) - (Z_1 - Z_2)f'(Z_2)| \leq \frac{\varepsilon |Z_1 - Z_2|}{1 + l^2}$$

ist, falls die in  $G$  liegenden Punkte  $Z_1$  und  $Z_2$  der Beziehung  $0 \leq |Z_1 - Z_2| < d$  genügen. Wählt man nun ein solches ganzes  $n$ , dass  $l < 2^n d$  ist, so gilt auf der  $n$ -ten Stufe für  $(\zeta_1 \zeta_2 \zeta_3)$ , falls  $\zeta_M = \frac{1}{3}(\zeta_1 + \zeta_2 + \zeta_3)$  gesetzt wird:

$$\left| \int_{\zeta_1 \zeta_2 \zeta_3} \{f(z) - f(\zeta_M) - (z - \zeta_M)f'(\zeta_M)\} dz \right| < \frac{\varepsilon}{4^n}.$$

Folglich ist nach Satz 8c, e, f:  $|I(\zeta_1 \zeta_2 \zeta_3)| = \left| \int_{\zeta_1 \zeta_2 \zeta_3} f(z) dz \right| < \frac{\varepsilon}{4^n}$ , mithin:

$$|I(z_1 z_2 z_3)| \leq \sum |I(\zeta_1 \zeta_2 \zeta_3)| < \varepsilon.$$

<sup>2)</sup> Proc. Kon. Akad. v. Wetensch., Amsterdam, 27, 189 (1924).



*Zweiter Fall.*  $L$  besteht aus  $n$  Strecken  $z_1 z_2, z_2 z_3, \dots, z_{n-1} z_n, z_n z_1$ . Man führt diesen Fall auf den vorigen zurück, indem man das Integral längs  $L$  darstellt als Summe von Integralen längs in  $G$  liegender Wege ( $z_a z_b z_c$ ).

*Allgemeiner Fall.* Zu vorgegebenem  $\varepsilon > 0$  bestimmt man  $d > 0$  derart, dass  $|f(z_1) - f(z_2)| < \frac{\varepsilon}{2l+1}$  ( $l = \text{Länge von } L$ ) bleibt, falls  $z_1$  und  $z_2$  der Bedingung  $|z_1 - z_2| < d$  genügen. Wählt man nun eine solche  $t$ -Zerlegung von  $L$ , dass:

a. jede Strecke  $z(t_{i+1}) z(t_i)$  in  $G$  liegt,

b.  $|z(t_{i+1}) - z(t_i)| < d$  ist und

$$c. \left| \int_L f(z) dz - \sum f(\xi) \{z(t_{i+1}) - z(t_i)\} \right| < \frac{1}{2} \varepsilon$$

ist, so gilt offenbar:

$$|I\{z(t_1), z(t_2), \dots, z(t_n)\} - \sum f(\xi) \{z(t_{i+1}) - z(t_i)\}| < \frac{1}{2} \varepsilon$$

also wegen  $I\{z(t_1), z(t_2), \dots, z(t_n)\} = 0$  und c:  $\left| \int_L f(z) dz \right| < \varepsilon$ .

**Satz 9.** Es sei  $f(z)$  regulär in einem einfach-zusammenhängenden Gebiete  $G$ . Weiter sei  $z_0$  ein fester,  $Z$  ein beliebiger innerer Punkt von  $G$  und  $L$  ein innerhalb  $G$  liegender Weg, der  $z_0$  und  $Z$  verbindet. Alsdann ist:

$$a) \quad F(Z) = \int_{z_0}^{L, Z} f(z) dz \quad \text{unabhängig von } L,$$

b)  $F(Z)$  regulär und c)  $F'(Z) = f(Z)$ .

*Beweis.* Nach dem Hauptsatz ist

$$\int_{z_0}^{L_1 Z} f(z) dz - \int_{z_0}^{L_2 Z} f(z) dz = 0.$$

Wählt man weiter bei vorgegebenem  $\varepsilon > 0$  die positive Zahl  $d$  derart, dass  $|f(z_1) - f(z_2)| < \varepsilon$  bleibt, sobald  $|z_1 - z_2| < 2d$  ist, so gilt:

$$\left| \frac{F(Z_3) - F(Z_2)}{Z_3 - Z_2} - f(Z_1) \right| < \varepsilon,$$

falls  $Z_1, Z_2$  und  $Z_3$  den Bedingungen  $0 < |Z_2 - Z_3| < d$  und

$0 < |Z_1 - Z_2| < d$  genügen, wie sich sofort aus

$$\left| \int_{Z_2}^{Z_3} [f(z) - f(Z_1)] dz \right| < \varepsilon |Z_3 - Z_2|$$

ergibt. Mithin ist  $F(z)$  nach Satz 3 regulär.

**Satz 10a.** *Es sei  $L$  der Weg*

$$z(t) = \frac{1 - t^2 + 2t\sqrt{-1}}{1 + t^2}, \quad 0 \leq t \leq a,$$

*also ein Bogen des Einheitskreises und es sei  $l$  die Länge von  $L$ .*

*Alsdann gilt:*  $\int_L \frac{dz}{z} = l\sqrt{-1}.$

*Beweis.* Offenbar ist:

$$\begin{aligned} \int_L \frac{dz}{z} &= \lim \sum \frac{z_{i+1} - z_i}{\sqrt{z_i z_{i+1}}} = \lim \sum \sqrt{-1} (z_{i+1} - z_i) \left( \frac{1}{z_{i+1}} - \frac{1}{z_i} \right) \\ &= \lim \sum \sqrt{-1} |z_{i+1} - z_i|^2 = l\sqrt{-1}. \end{aligned}$$

**Satz 10b.** *Liegen alle Punkte eines geschlossenen Weges  $L$  auf einem Kreis  $|z - z_0| = R$ , so gilt:*  $\int_L \frac{dz}{z - z_0} = 2k\pi\sqrt{-1}$ , *wo  $2\pi$  den Umfang des Einheitskreises bezeichnet und  $k$  eine ganze positive oder negative Zahl oder Null ist.*

*Beweis.* Für den Fall  $z_0 = 0$  und  $R = 1$  folgt der Satz aus 10a. Der allgemeine Fall lässt sich sofort auf diesen besonderen Fall zurückführen.

**Satz 10c.** *Liegt ein geschlossener Weg  $L$  ausserhalb eines Kreises mit dem Mittelpunkt  $Z$ , so gilt:*  $\int_L \frac{dz}{z - Z} = 2k\pi\sqrt{-1}.$

*Ist  $k > 0$  bzw.  $k = 0$ , so sagt man, dass  $L$  den Punkt  $Z$   $k$ -mal im positiven Sinne bzw. nicht umschliesst.*

Wie man leicht einsieht, kommt diese Definition für positives  $k$  (die Fälle  $k$  negativ und  $k = 0$  gehen analog) darauf hinaus, dass wenn  $z$  den Weg  $L$  durchläuft, der Radiusvector von  $z$  aus  $Z$  eine Bahn beschreibt, welche sich stetig in eine solche überführen lässt, die  $k$  Male der Reihe nach die rechten Winkel zwischen den Richtungen  $+1$  und  $+i$ , zwischen  $+i$  und  $-1$ , zwischen  $-1$  und  $-i$  und zwischen  $-i$  und  $+1$  beschreibt.

*Beweis.* Der allgemeine Fall lässt sich auf den Fall  $Z = 0$  zurück-

führen und diesen beweist man, indem man den Weg  $L$  zerlegt in Teilwege  $z_1 z_2, z_2 z_3, \dots, z_{n-1} z_n, z_n z_1$ , derart dass jeder Teilweg in wenigstens einer der vier Halbebenen  $\pm \Re(z) \geq 0, \pm \Im(z) \geq 0$  liegt. Sei  $L^*$  der Weg  $\frac{z(t)}{|z(t)|}$  und  $z_i^* = \frac{z_i}{|z_i|}$ . Es gilt dann:

$$\int_{z_i z_{i+1}}^L \frac{dz}{z} - \int_{z_i^* z_{i+1}^*}^{L^*} \frac{dz}{z} = \sum \int_{z_i z_{i+1} z_{i+1}^* z_i^*} \frac{dz}{z} = 0,$$

denn der geschlossene Weg  $z_i z_{i+1} z_{i+1}^* z_i^*$ , der sich aus einem Teilweg von  $L$  bzw.  $L^*$  und den Strecken  $z_{i+1} z_{i+1}^*$  und  $z_i^* z_i$  zusammensetzt, liegt in einem einfach-zusammenhängenden Gebiete in welchem die Funktion  $\frac{1}{z}$  regulär ist. Also ist:  $\int_{z_i z_{i+1}}^L \frac{dz}{z} = \int_{z_i^* z_{i+1}^*}^{L^*} \frac{dz}{z}$  und da die Punkte von  $L^*$  auf dem Kreis  $|z|=1$  liegen, folgt die Behauptung aus Satz 10b.

*Bemerkung.* Im folgenden treten Integrale  $I = \int_G f(z) dz$  auf, wo  $G$  ein einfach-zusammenhängendes Gebiet ist, dessen Rand aus den Punkten eines geschlossenen Weges besteht, der die inneren Punkte von  $G$  einmal im positiven Sinne umschliesst. Mit  $I$  ist dann immer das über diesen geschlossenen Weg erstreckte Integral gemeint. Weiter sagen wir, dass ein geschlossener Weg  $L$  ein Gebiet  $G$   $k$ -mal umschliesst, falls es eine solche positive Zahl  $a$  gibt, dass jeder Punkt von  $L$  und jeder Punkt von  $G$  um mehr als  $a$  von einander entfernt sind<sup>3)</sup> und der Weg  $L$  jeden Punkt von  $G$   $k$ -mal umschliesst. Letzteres ist der Fall, wenn  $L$  einen Punkt von  $G$   $k$ -mal umschliesst, wie sich aus der Stetigkeit nach  $Z$  von  $\frac{1}{z-Z}$  für  $z \neq Z$  folgern lässt.

**Satz 11.** *Es liege der Kreis  $K \equiv |z - z_0| = R$  in einem einfach-zusammenhängenden Gebiete  $G$  und es sei  $f(z)$  in  $G$  ausserhalb  $K$  regulär. Weiter seien  $L_1$  und  $L_2$  zwei in  $G$  liegende, geschlossene Wege, die den Punkt  $z_0$   $k$ -mal umschliessen und ausserhalb des Kreises  $K_a \equiv |z - z_0| = R + a$  ( $a > 0$ ) liegen. Alsdann gilt:*

$$\int_{L_1} f(z) dz = \int_{L_2} f(z) dz.$$

*Beweis.* Ohne Beschränkung der Allgemeinheit sei  $z_0 = 0$ . Wir setzen für  $i = 1, 2$ :  $L_i = z^i(t)$ ,  $z^i(t) = \frac{z^i(t)}{|z^i(t)|} (R + a)$  und  $L_i^* = z^i(t)$ . Zerlegt man

<sup>3)</sup> Nach einem BROUWERSchen Satz (sich Fussnote 2, Seite 177) ist hierfür die Bedingung, dass jeder Punkt von  $L$  eine positive Entfernung von  $G$  hat, hinreichend.

nun  $L$  in Teilwege  $z_1^i z_2^i, z_2^i z_3^i, \dots, z_n^i z_1^i$ , derart, dass jeder Teilweg in wenigstens einer der vier Halbebenen  $\pm \Re(z) > 0$ ,  $\pm \Im(z) > 0$  liegt und setzt man noch  $z_j^i = \frac{z_j^i}{|z_j^i|} (R + a)$ , so gilt:

$$\int_{L_i} f(z) dz - \int_{L_i^*} f(z) dz = \sum \int_{z_j^i z_{j+1}^i z_{j+1}^{*i} z_j^{*i}} f(z) dz = 0, \quad \dots \quad (A)$$

denn der geschlossene Weg  $z_j^i z_{j+1}^i z_{j+1}^{*i} z_j^{*i}$ , der sich aus Teilwegen von  $L_i$  und  $L_i^*$  und den beiden Strecken  $z_{j+1}^i z_{j+1}^{*i}$  und  $z_j^i z_j^{*i}$  zusammensetzt, liegt in einem einfach-zusammenhängenden Gebiete, in welchem  $f(z)$  regulär ist. Es gilt weiter:

$$\int_{L_1^*} f(z) dz = \int_{L_2^*} f(z) dz, \quad \dots \quad (B)$$

denn beide Integrale sind über den Kreis  $K_a$  zu erstrecken und dabei wird dieser Kreis wegen:

$$\int_{L_i} \frac{dz}{z} = \int_{L_i^*} \frac{dz}{z} = 2\pi k \sqrt{-1}$$

genau  $k$ -mal durchlaufen. Aus (A) und (B) folgt die Behauptung.

*CAUCHYSche Integralformel.* Es sei  $f(z)$  regulär in einem einfach-zusammenhängenden Gebiete  $G$  und es sei  $L$  ein geschlossener Weg, der in  $G$  liegt und der den Punkt  $z_0$  einmal im positiven Sinne umschliesst. Alsdann ist:

$$\int_L \frac{f(z) dz}{z - z_0} = 2\pi \sqrt{-1} \cdot f(z_0).$$

*Beweis.* Bestimmt man bei vorgegebenem  $\varepsilon > 0$  eine positive Zahl  $r$  derart, dass der Kreis  $|z - z_0| = r$  in  $G$  liegt und auf seinem Rande

$|f(z) - f(z_0)| < \frac{\varepsilon}{2\pi}$  ist, so gilt:

$$\int_{|z-z_0|=r} \frac{f(z) - f(z_0)}{z - z_0} dz < \varepsilon, \text{ also nach Satz 10b: } \left| \int_{|z-z_0|=r} \frac{f(z) dz}{z - z_0} - 2\pi \sqrt{-1} f(z_0) \right| < \varepsilon. \quad (A)$$

Nach dem vorigen Satz ist:

$$\int_L \frac{f(z) dz}{z - z_0} = \int_{|z-z_0|=r} \frac{f(z) dz}{z - z_0} \quad \dots \quad (B)$$

Aus (A) und (B) folgt die Behauptung.



**Satz 12.** Es sei  $L$  ein geschlossener Weg, der ein einfach-zusammenhängendes Gebiet  $G$  einmal im positiven Sinne umschliesst und es sei die Funktion  $f(z)$  gleichmässig stetig längs  $L$ . Alsdann ist die Funktion:

$$\varphi(Z) = \frac{1}{2\pi\sqrt{-1}} \int_L^Z \frac{f(z) dz}{z-Z}$$

in  $G$  regulär und es gilt:

$$\varphi^n(Z) = \frac{n!}{2\pi\sqrt{-1}} \int_L^Z \frac{f(z) dz}{(z-Z)^{n+1}}.$$

*Beweis:* Sei  $f(z)$  längs  $L$  dem absoluten Betrage nach kleiner als die positive Zahl  $M$  und sei  $l$  die Länge von  $L$ . Setzt man für  $n=0, 1, 2, \dots$ :

$$V_n(z z_1 z_2 z_3) = \frac{n!}{z_2 - z_3} \left[ \frac{1}{(z - z_2)^{n+1}} - \frac{1}{(z - z_3)^{n+1}} \right] - \frac{(n+1)!}{(z - z_1)^{n+2}},$$

so lässt sich bei vorgegebenem  $\varepsilon > 0$  für jedes  $n$  eine positive Zahl  $d_n$  so bestimmen, dass  $|V_n(z z_1 z_2 z_3)| < \frac{2\varepsilon\pi}{Ml}$  bleibt, falls die in  $G$  liegenden Punkte  $z_1 z_2 z_3$  den Bedingungen  $0 < |z_1 - z_2| < d_n$  und  $0 < |z_1 - z_3| < d_n$  genügen und der Punkt  $z$  auf  $L$  liegt. Setzt man:

$$g_n(Z) = \frac{n!}{2\pi\sqrt{-1}} \int_L^Z \frac{f(z) dz}{(z-Z)^{n+1}},$$

so ist also:

$$\left| \frac{g_n(z_2) - g_n(z_3)}{z_2 - z_3} - g_{n+1}(z_1) \right| = \left| \frac{1}{2\pi\sqrt{-1}} \int_L^Z f(z) V_n(z z_1 z_2 z_3) dz \right| < \varepsilon.$$

Für  $n=0$  folgt hieraus nach der Umkehrung von Satz 3 § 1, dass  $\varphi(Z) = g_0(Z)$  in  $G$  regulär ist. Wiederholte Anwendung für  $n=1, 2, \dots$  usw. ergibt die zweite Behauptung.

**Satz 13.** Es sei  $f(z)$  in einem einfach-zusammenhängenden Gebiete  $G$  regulär. Es sei  $L$  ein geschlossener Weg in  $G$ , der ein einfach-zusammenhängendes Gebiet  $G^*$  einmal im positiven Sinne umschliesst. Alsdann ist die Ableitung  $f'(z)$  in  $G^*$  regulär und hat  $f(z)$  in  $G^*$  Ableitungen beliebiger Ordnung, die alle in  $G^*$  regulär sind.

*Beweis.* Folgt unmittelbar aus den beiden vorigen Sätzen.

**Satz 14.** Es sei  $f(z)$  regulär in dem Ring  $r_1 \leq |z - z_0| \leq r_2$  ( $R$ ),  $Z$  ein Punkt von  $R$  und es seien  $C_1$  und  $C_2$  zwei solche in  $R$  liegende

Kreise um  $z_0$ , dass  $Z$  innerhalb  $C_2$  doch ausserhalb  $C_1$  liegt. Alsdann gilt:

$$2\pi\sqrt{-1}f(Z) = \int_{C_2} \frac{f(z)dz}{z-Z} - \int_{C_1} \frac{f(z)dz}{z-Z},$$

falls die Kreise im positiven Sinne durchlaufen werden.

*Beweis.* Sei  $C_3$  ein Kreis um  $Z$ , der innerhalb  $C_2$  jedoch ausserhalb  $C_1$  liegt. Verbindet man  $C_1$ ,  $C_2$  und  $C_3$  durch Hilfswege, so ergibt sich:

$$\int_{C_3} \frac{f(z)dz}{z-Z} = \int_{C_1} \frac{f(z)dz}{z-Z} + \int_{C_2} \frac{f(z)dz}{z-Z}. \quad \dots \dots (A)$$

Nach der CAUCHYSchen Integralformel ist:

$$\int_{C_3} \frac{f(z)dz}{z-Z} = 2\pi\sqrt{-1}f(Z). \quad \dots \dots (B)$$

Aus (A) und (B) folgt die Behauptung.

§ 4. Im folgenden ist mit konvergent immer positiv konvergent gemeint.

**Satz 15.** *Es seien die Funktionen  $f_n(z)$  in einem Gebiete  $G$  regulär und es sei dort  $|f_n(z)| < a_n$ . Falls nun die Reihe  $\sum a_n$  konvergiert, so konvergiert die Reihe  $\sum f_n(z)$  gleichmässig in  $G$ .*

**Satz 16a.** *Es konvergiere die Reihe  $\sum f_n(z)$  gleichmässig in  $G$  und es sei dort jede Funktion  $f_n(z)$  gleichmässig stetig. Alsdann ist die Summe  $f(z)$  der Reihe gleichmässig stetig in  $G$ .*

**Satz 16b.** *Unter den Voraussetzungen von Satz 16a konvergiert die Reihe  $\sum \int_L f_n(z) dz$  zu der Summe*

$$\int_L \sum f_n(z) dz = \int_L f(z) dz.$$

**Satz 16c.** (WEIERSTRASSscher Doppelreihensatz). *Es konvergiere die Reihe  $\sum f_n(z)$  gleichmässig in einem einfach-zusammenhängenden Gebiete  $G$  und es sei dort jede Funktion  $f_n(z)$  regulär. Weiter sei  $L$  ein in  $G$  liegender, geschlossener Weg, der ein einfach-zusammenhängendes Gebiet  $G^*$  einmal im positiven Sinne umschliesst. Alsdann ist die Summe  $f(z)$  der Reihe  $\sum f_n(z)$  in  $G^*$  regulär. Es ist weiter die Reihe  $\sum f_n^{(p)}(z)$  in  $G^*$  gleichmässig konvergent und ihre Summe der  $p$ -ten Ableitung von  $f(z)$  gleich.*

*Beweis.* Sei  $Z$  ein Punkt von  $G^*$ . Nach der CAUCHYSchen Integralformel ist:

$$f(Z) = \sum f_n(Z) = \sum \frac{1}{2\pi\sqrt{-1}} \int_L \frac{f_n(z) dz}{z-Z} \dots \dots \dots (A)$$

Nach Satz 16b gilt:

$$\sum \int_L \frac{f_n(z) dz}{z-Z} = \int_L \frac{\sum f_n(z)}{z-Z} dz = \int_L \frac{f(z) dz}{z-Z} \dots \dots \dots (B)$$

Aus (A) und (B) folgt:

$$f(Z) = \frac{1}{2\pi\sqrt{-1}} \int_L \frac{f(z) dz}{z-Z}.$$

Also ist  $f(Z)$  nach Satz 12 regulär in  $G^*$ . Es habe nun jeder Punkt von  $G^*$  zu jedem Punkt von  $L$  eine Entfernung  $> a > 0$  und es sei  $l$  die Länge von  $L$ . Bestimmt man dann zu vorgegebenem  $\varepsilon > 0$   $N$  derart, dass die Ungleichung:

$$\left| \sum_{n+1}^{n+q} f_i(z) \right| < \frac{\varepsilon a^{p+1}}{l p!}$$

in  $G$  für jedes  $n > N$  gilt, so ist:

$$\left| \sum_{n+1}^{n+q} f_i^p(Z) \right| = \frac{p!}{2\pi} \left| \sum_{n+1}^{n+q} \int_L \frac{f_i(z) dz}{(z-Z)^{p+1}} \right| < \varepsilon.$$

Folglich ist  $\sum f_i^p(z)$  in  $G^*$  gleichmässig konvergent. Schliesslich ist:

$$f^p(Z) = \frac{p!}{2\pi\sqrt{-1}} \int_L \frac{f(z) dz}{(z-Z)^{p+1}} = \sum \frac{p!}{2\pi\sqrt{-1}} \int_L \frac{f_n(z) dz}{(z-Z)^{p+1}} = \sum f_n^p(Z).$$

**Satz 17.** Es sei  $f(z)$  regulär in einem einfach-zusammenhängenden Gebiete  $G$  und es liege der Kreis  $|z - z_0| = R$  (C) in dem Innern von  $G$ . Es sei weiter  $L$  ein in  $G$  liegender geschlossener Weg, der den Punkt  $z_0$  einmal im positiven Sinne umschliesst. Ist nun  $Z$  ein innerer

Punkt von  $C$ , so konvergiert die Reihe  $\sum a_n (Z - z_0)^n$ , wo  $a_n = \int_L \frac{f(z) dz}{(z - z_0)^{n+1}}$

ist, gegen die Summe  $f(Z)$ . Es gilt weiter:  $a_n = \frac{1}{n!} f^n(z_0)$  (TAYLORSche Entwicklung).

*Beweis.* Nach der CAUCHYSchen Integralformel, Satz 16b und Satz 11 ist:

$$\begin{aligned} f(Z) &= \frac{1}{2\pi\sqrt{-1}} \int_C \frac{f(z) dz}{z-Z} = \frac{1}{2\pi\sqrt{-1}} \int_C f(z) dz \sum_0^{\infty} \frac{(Z-z_0)^n}{(z-z_0)^{n+1}} \\ &= \sum_0^{\infty} (Z-z_0)^n \cdot \frac{1}{2\pi\sqrt{-1}} \int_C \frac{f(z) dz}{(z-z_0)^{n+1}} = \sum_0^{\infty} a_n (Z-z_0)^n. \end{aligned}$$

Dass  $a_n = \frac{1}{n!} f^n(z_0)$  ist, folgt aus Satz 16c.

**Satz 18.** Es sei  $f(z)$  eine in dem Ring  $r_1 \leq |z-z_0| \leq r_2$  ( $R$ ) reguläre Funktion und es sei  $L$  ein in  $R$  liegender geschlossener Weg, der den Punkt  $z_0$  einmal im positiven Sinne umschliesst. Alsdann gilt für jedes  $Z$ , das der Bedingung  $r_1 < |Z-z_0| < r_2$  genügt:

$$f(Z) = \sum_0^{\infty} a_n (Z-z_0)^n + \sum_1^{\infty} b_n (Z-z_0)^{-n},$$

wo  $a_n = \frac{1}{2\pi\sqrt{-1}} \int_L \frac{f(z) dz}{(z-z_0)^{n+1}}$  und  $b_n = \frac{1}{2\pi\sqrt{-1}} \int_L f(z) (z-z_0)^{n-1} dz$  ist.

(LAURENTSche Entwicklung.)

*Beweis.* Seien  $C_1$  und  $C_2$  zwei solche in  $R$  liegende Kreise um  $z_0$ , dass der Punkt  $Z$  innerhalb  $C_2$  jedoch ausserhalb  $C_1$  liegt. Nach Satz 14, 16b und 11 gilt nun:

$$\begin{aligned} 2\pi\sqrt{-1} f(Z) &= \int_{C_2} \frac{f(z) dz}{z-Z} - \int_{C_1} \frac{f(z) dz}{z-Z} \\ &= \int_{C_2} \sum_0^{\infty} \frac{(Z-z_0)^n}{(z-z_0)^{n+1}} f(z) dz + \int_{C_1} \sum_1^{\infty} (Z-z_0)^{-n} (z-z_0)^{n-1} f(z) dz \\ &= \sum_0^{\infty} (Z-z_0)^n \int_{C_2} \frac{f(z) dz}{(z-z_0)^{n+1}} + \sum_1^{\infty} (Z-z_0)^{-n} \int_{C_1} f(z) (z-z_0)^{n-1} dz \\ &= 2\pi\sqrt{-1} \sum_0^{\infty} a_n (Z-z_0)^n + 2\pi\sqrt{-1} \sum_1^{\infty} b_n (Z-z_0)^{-n}. \end{aligned}$$



**Mathematics.** — *Neue Integraldarstellungen für WHITTAKERSche Funktionen.* (Zweite Mitteilung). Von C. S. MEIJER. (Communicated by Prof. J. G. VAN DER CORPUT.)

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§ 4. Mit Hilfe der Formeln (17) und (49) der Sätze 1 bzw. 2 kann man ohne Mühe zahlreiche wichtige bestimmte Integrale berechnen; denn sehr viele häufig in der Analyse auftretenden Funktionen sind nur Spezialfälle<sup>19)</sup> der Funktion  $G_{p,q}^{m,n}(w)$ .

In einigen früheren Arbeiten<sup>20)</sup> habe ich schon verschiedene Sonderfälle der Formeln (17) und (49) ausführlich untersucht. Das Ziel der vorliegenden Abhandlung ist mit Hilfe von (17) und (49) Integraldarstellungen abzuleiten für die WHITTAKERSchen Funktionen  $M_{k,m}(\zeta)$  und  $W_{k,m}(\zeta)$ . Diese Funktionen können folgenderweise definiert werden<sup>21)</sup>:

$$e^{\frac{1}{2}\zeta} M_{k,m}(\zeta) = \zeta^{\frac{1}{2}+m} {}_1F_1\left(\frac{1}{2}-k+m; 1+2m; \zeta\right), \quad \dots \quad (53)$$

$$e^{\frac{1}{2}\zeta} W_{k,m}(\zeta) = \left. \begin{aligned} & \frac{\Gamma(-2m)}{\Gamma(\frac{1}{2}-k-m)} \zeta^{\frac{1}{2}+m} {}_1F_1\left(\frac{1}{2}-k+m; 1+2m; \zeta\right) \\ & + \frac{\Gamma(2m)}{\Gamma(\frac{1}{2}-k+m)} \zeta^{\frac{1}{2}-m} {}_1F_1\left(\frac{1}{2}-k-m; 1-2m; \zeta\right) \end{aligned} \right\}. \quad (54)$$

Nun ist<sup>22)</sup>

$$e^{-\zeta} {}_1F_1(\alpha; \beta; \zeta) = {}_1F_1(\beta-\alpha; \beta; -\zeta); \quad \dots \quad (55)$$

für die Funktionen  $M_{k,m}(\zeta)$  und  $W_{k,m}(\zeta)$  gilt also auch

$$e^{-\frac{1}{2}\zeta} M_{k,m}(\zeta) = \zeta^{\frac{1}{2}+m} {}_1F_1\left(\frac{1}{2}+k+m; 1+2m; -\zeta\right), \quad \dots \quad (56)$$

$$e^{-\frac{1}{2}\zeta} W_{k,m}(\zeta) = \left. \begin{aligned} & \frac{\Gamma(-2m)}{\Gamma(\frac{1}{2}-k-m)} \zeta^{\frac{1}{2}+m} {}_1F_1\left(\frac{1}{2}+k+m; 1+2m; -\zeta\right) \\ & + \frac{\Gamma(2m)}{\Gamma(\frac{1}{2}-k+m)} \zeta^{\frac{1}{2}-m} {}_1F_1\left(\frac{1}{2}+k-m; 1-2m; -\zeta\right) \end{aligned} \right\}. \quad (57)$$

Aus (1), (53), (56), (54) und (57) geht jetzt hervor

<sup>19)</sup> Man vergl. das Verzeichnis in § 5. Siehe auch (58), (59), (60) und (61).

<sup>20)</sup> Man vergl. z.B. [12] und [19]; siehe auch [17], Formel (1).

<sup>21)</sup> WHITTAKER and WATSON, [27], §§ 16.1 und 16.41.

<sup>22)</sup> WATSON, [26], 102; WHITTAKER and WATSON, [27], § 16.11. Für einen sehr einfachen Beweis von (55) siehe man MACROBERT, [10], 83.

$$e^{\frac{1}{2}\zeta} M_{k,m}(\zeta) = \zeta^{\frac{1}{2}} \Gamma(1+2m) \Gamma(\tfrac{1}{2}+k-m) G_{1,2}^{1,0} \left( \zeta \left| \begin{array}{c} \frac{1}{2}+k \\ m, -m \end{array} \right. \right), \quad (58)$$

$$e^{-\frac{1}{2}\zeta} M_{k,m}(\zeta) = \frac{\zeta^{\frac{1}{2}} \Gamma(1+2m)}{\Gamma(\tfrac{1}{2}+k+m)} G_{1,2}^{1,1} \left( \zeta \left| \begin{array}{c} \frac{1}{2}-k \\ m, -m \end{array} \right. \right), \quad \dots \quad (59)$$

$$e^{\frac{1}{2}\zeta} W_{k,m}(\zeta) = \frac{\zeta^{\frac{1}{2}}}{\Gamma(\tfrac{1}{2}-k+m) \Gamma(\tfrac{1}{2}-k-m)} G_{1,2}^{2,1} \left( \zeta \left| \begin{array}{c} \frac{1}{2}+k \\ m, -m \end{array} \right. \right), \quad (60)$$

$$e^{-\frac{1}{2}\zeta} W_{k,m}(\zeta) = \zeta^{\frac{1}{2}} G_{1,2}^{2,0} \left( \zeta \left| \begin{array}{c} \frac{1}{2}-k \\ m, -m \end{array} \right. \right). \quad \dots \quad (61)$$

Man kann nun leicht die folgenden Sätze beweisen:

**Satz 3.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \frac{3}{2}\pi$ , so besitzt die Funktion  $W_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{-\frac{1}{2}\zeta} \Gamma(\tfrac{1}{2}-\kappa+\lambda) \Gamma(\tfrac{1}{2}-\kappa-\lambda)}{\Gamma(\tfrac{1}{2}-k+m) \Gamma(\tfrac{1}{2}-k-m)} \\ &\times \int_0^{\infty} e^{-i \arg \zeta} v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} W_{\kappa,\lambda}(v) G_{2,4}^{2,1} \left( \zeta v \left| \begin{array}{c} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ m, -m, \beta+\lambda, \beta-\lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (62)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit <sup>23)</sup>

$$\Re(1 \pm m - \beta \pm \lambda) > 0, \quad \Re(\beta - k - \kappa) > 0, \quad \Re(\tfrac{1}{2} + k + \beta - 3\kappa) > 0.$$

**Satz 4.** Die Funktion  $W_{k,m}(\zeta)$  besitzt für jedes  $\zeta \neq 0$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{-\frac{1}{2}\zeta}}{\Gamma(\tfrac{1}{2}-k+m) \Gamma(\tfrac{1}{2}-k-m)} \\ &\times \int_0^{\infty} v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} W_{\kappa,\lambda}(v) G_{2,4}^{2,2} \left( \zeta v \left| \begin{array}{c} \beta+\kappa-\frac{1}{2}, \frac{1}{2}+k \\ m, -m, \beta+\lambda, \beta-\lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (63)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit  $\Re(1 \pm m - \beta \pm \lambda) > 0$ .

<sup>23)</sup>  $\Re(1 \pm m - \beta \pm \lambda) > 0$  bedeutet  $\Re(1+m-\beta+\lambda) > 0$ ,  $\Re(1+m-\beta-\lambda) > 0$ ,  $\Re(1-m-\beta+\lambda) > 0$  und  $\Re(1-m-\beta-\lambda) > 0$ .

Damit alle in (62) vorkommenden Funktionen einen Sinn haben, muss ich überdies noch annehmen (man vergl. auch (3) und (4))

$$\tfrac{1}{2} - \kappa \pm \lambda \neq 0, -1, -2, \dots, \quad 2m \neq 0, \pm 1, \pm 2, \dots, \quad \tfrac{1}{2} - k \pm m \neq 0, -1, -2, \dots$$

Bedingungen derartiger Gestalt werden aber in der Folge bei der Fassung der Sätze und bei den Spezialfällen nicht notiert; der Kürze halber werden nur die Konvergenzbedingungen angeschrieben.

**Satz 5.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \frac{3}{2}\pi$ , so besitzt die Funktion  $W_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{-\frac{1}{2}\zeta} \Gamma(\frac{1}{2} + \kappa + \lambda)}{\Gamma(\frac{1}{2} - k + m) \Gamma(\frac{1}{2} - k - m) \Gamma(1 + 2\lambda)} \\ &\times \int_0^{\infty e^{i\psi}} v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} M_{\kappa,\lambda}(v) G_{2,4}^{3,1} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} + k, \beta + \kappa - \frac{1}{2} \\ \beta + \lambda, m, -m, \beta - \lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (64)$$

Hierin ist

$$\text{Max}(-\frac{1}{2}\pi, -\pi - \arg \zeta) < \psi < \text{Min}(\frac{1}{2}\pi, \pi - \arg \zeta); \quad . \quad . \quad (65)$$

$\beta, \kappa$  und  $\lambda$  sind beliebig mit

$$\Re(\frac{1}{2} + \lambda) > 0, \quad \Re(1 \pm m - \beta + \lambda) > 0, \quad \Re(\beta - k + \kappa) > 0.$$

**Satz 6.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \frac{3}{2}\pi$ , so besitzt die Funktion  $W_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{-\frac{1}{2}\zeta} \Gamma(\frac{1}{2} - \kappa + \lambda)}{2\pi i \Gamma(\frac{1}{2} - k + m) \Gamma(\frac{1}{2} - k - m) \Gamma(1 + 2\lambda)} \\ &\times \int_{-\infty e^{i\psi}}^{(0+)} v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} M_{\kappa,\lambda}(v) G_{2,4}^{4,1} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} + k, \beta - \kappa - \frac{1}{2} \\ \beta + \lambda, \beta - \lambda, m, -m \end{array} \right. \right) dv. \end{aligned} \right\} \quad (66)$$

Hierin genügt  $\psi$  der Bedingung (65);  $\beta, \kappa$  und  $\lambda$  sind beliebig mit  $\Re(\beta - k - \kappa) > 0$ .

**Satz 7.** Die Funktion  $W_{k,m}(\zeta)$  besitzt für jedes  $\zeta \neq 0$  die Integraldarstellung

$$W_{k,m}(\zeta) = \zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \int_0^{\infty} v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} W_{\kappa,\lambda}(v) G_{2,4}^{2,1} \left( \zeta v \left| \begin{array}{c} \beta + \kappa - \frac{1}{2}, \frac{1}{2} - k \\ m, -m, \beta + \lambda, \beta - \lambda \end{array} \right. \right) dv. \quad (67)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit  $\Re(1 \pm m - \beta \pm \lambda) > 0$ .

**Satz 8.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \frac{1}{2}\pi$ , so besitzt die Funktion  $W_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(\frac{1}{2} + \kappa + \lambda)}{\Gamma(1 + 2\lambda)} \\ &\times \int_0^{\infty e^{-i\arg \zeta}} v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} M_{\kappa,\lambda}(v) G_{2,4}^{3,0} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} - k, \beta + \kappa - \frac{1}{2} \\ \beta + \lambda, m, -m, \beta - \lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (68)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit

$$\Re(\tfrac{1}{2} + \lambda) > 0, \quad \Re(1 \pm m - \beta + \lambda) > 0, \quad \Re(\tfrac{1}{2} - k + \beta + 3\kappa) > 0.$$

**Satz 9.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \tfrac{1}{2}\pi$ , so besitzt die Funktion  $W_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} W_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(\tfrac{1}{2} - \kappa + \lambda)}{2\pi i \Gamma(1 + 2\lambda)} \\ &\times \int_{-\infty e^{-i \arg \zeta}}^{(0+)} v^{-\beta - \frac{1}{2}} e^{\frac{1}{2}v} M_{\kappa, \lambda}(v) G_{2,4}^{4,0} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} - k, \beta - \kappa - \frac{1}{2} \\ \beta + \lambda, \beta - \lambda, m, -m \end{array} \right. \right) dv. \end{aligned} \right\} \quad (69)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit  $\Re(\tfrac{1}{2} - k + \beta - 3\kappa) > 0$ .

**Satz 10.** Die Funktion  $M_{k,m}(\zeta)$  besitzt für jedes  $\zeta \neq 0$  die Integraldarstellung

$$\left. \begin{aligned} M_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(1 + 2m)}{\Gamma(\tfrac{1}{2} + k + m)} \\ &\times \int_0^{\infty} v^{-\beta - \frac{1}{2}} e^{-\frac{1}{2}v} W_{\kappa, \lambda}(v) G_{2,4}^{1,2} \left( \zeta v \left| \begin{array}{c} \beta + \kappa - \frac{1}{2}, \frac{1}{2} - k \\ m, -m, \beta + \lambda, \beta - \lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (70)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit  $\Re(1 + m - \beta \pm \lambda) > 0$ .

**Satz 11.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \tfrac{1}{2}\pi$ , so besitzt die Funktion  $M_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} M_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(1 + 2m) \Gamma(\tfrac{1}{2} + \kappa + \lambda)}{\Gamma(\tfrac{1}{2} + k + m) \Gamma(1 + 2\lambda)} \\ &\times \int_0^{\infty e^{-i \arg \zeta}} v^{-\beta - \frac{1}{2}} e^{-\frac{1}{2}v} M_{\kappa, \lambda}(v) G_{2,4}^{2,1} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} - k, \beta + \kappa - \frac{1}{2} \\ \beta + \lambda, m, -m, \beta - \lambda \end{array} \right. \right) dv. \end{aligned} \right\} \quad (71)$$

Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit

$$\Re(\tfrac{1}{2} + \lambda) > 0, \quad \Re(1 + m - \beta + \lambda) > 0, \quad \Re(\beta + k + \kappa) > 0, \quad \Re(\tfrac{1}{2} - k + \beta + 3\kappa) > 0.$$

**Satz 12.** Ist  $\zeta \neq 0$  und  $|\arg \zeta| < \tfrac{1}{2}\pi$ , so besitzt die Funktion  $M_{k,m}(\zeta)$  die Integraldarstellung

$$\left. \begin{aligned} M_{k,m}(\zeta) &= \frac{\zeta^{\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(1 + 2m) \Gamma(\tfrac{1}{2} - \kappa + \lambda)}{2\pi i \Gamma(\tfrac{1}{2} + k + m) \Gamma(1 + 2\lambda)} \\ &\times \int_{-\infty e^{-i \arg \zeta}}^{(0+)} v^{-\beta - \frac{1}{2}} e^{\frac{1}{2}v} M_{\kappa, \lambda}(v) G_{2,4}^{3,1} \left( \zeta v \left| \begin{array}{c} \frac{1}{2} - k, \beta - \kappa - \frac{1}{2} \\ \beta + \lambda, \beta - \lambda, m, -m \end{array} \right. \right) dv. \end{aligned} \right\} \quad (72)$$



Hierin sind  $\beta, \kappa$  und  $\lambda$  beliebig mit

$$\Re(\beta + k - \kappa) > 0, \quad \Re(\tfrac{1}{2} - k + \beta - 3\kappa) > 0.$$

Diese Sätze sind Spezialfälle der Sätze 1 bzw. 2. Aus der Definition der Funktion  $G_{p,q}^{m,n}(w)$  ergibt sich nämlich

$$G_{p+1,q+1}^{m+1,n} \left( w \left| \begin{matrix} a_1, \dots, a_p, \gamma \\ \gamma, b_1, \dots, b_q \end{matrix} \right. \right) = G_{p,q}^{m,n} \left( w \left| \begin{matrix} a_1, \dots, a_p \\ b_1, \dots, b_q \end{matrix} \right. \right) \quad (n \leq p; m \geq 1), \quad (73)$$

$$G_{p+1,q+1}^{m,n+1} \left( w \left| \begin{matrix} \delta, a_1, \dots, a_p \\ b_1, \dots, b_q, \delta \end{matrix} \right. \right) = G_{p,q}^{m,n} \left( w \left| \begin{matrix} a_1, \dots, a_p \\ b_1, \dots, b_q \end{matrix} \right. \right) \quad (m \leq q; n \geq 0) \quad (74)$$

und überdies

$$G_{p,q}^{m,n} \left( w \left| \begin{matrix} a_1 + t, \dots, a_p + t \\ b_1 + t, \dots, b_q + t \end{matrix} \right. \right) = w^t G_{p,q}^{m,n} \left( w \left| \begin{matrix} a_1, \dots, a_p \\ b_1, \dots, b_q \end{matrix} \right. \right). \quad (75)$$

Die Anwendung von (75) auf (59), (60) und (61) liefert

$$\begin{aligned} v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} M_{\kappa,\lambda}(v) &= \frac{\Gamma(1+2\lambda)}{\Gamma(\frac{1}{2}+\kappa+\lambda)} G_{1,2}^{1,1} \left( v \left| \begin{matrix} \frac{1}{2}-\kappa-\beta \\ \lambda-\beta, -\lambda-\beta \end{matrix} \right. \right), \\ v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} W_{\kappa,\lambda}(v) &= \frac{1}{\Gamma(\frac{1}{2}-\kappa+\lambda) \Gamma(\frac{1}{2}-\kappa-\lambda)} G_{1,2}^{2,1} \left( v \left| \begin{matrix} \frac{1}{2}+\kappa-\beta \\ \lambda-\beta, -\lambda-\beta \end{matrix} \right. \right), \\ v^{-\beta-\frac{1}{2}} e^{-\frac{1}{2}v} W_{\kappa,\lambda}(v) &= G_{1,2}^{2,0} \left( v \left| \begin{matrix} \frac{1}{2}-\kappa-\beta \\ \lambda-\beta, -\lambda-\beta \end{matrix} \right. \right). \end{aligned} \quad (76)$$

Aus (76), (17), (73) und (74) folgt nun (ich setze  $v = e^{-i \arg \zeta} x$  und benutze den vierten Fall von Satz 1)

$$\begin{aligned} & \Gamma(\tfrac{1}{2}-\kappa+\lambda) \Gamma(\tfrac{1}{2}-\kappa-\lambda) \int_0^{\infty e^{-i \arg \zeta}} v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} W_{\kappa,\lambda}(v) G_{2,4}^{2,1} \left( \zeta v \left| \begin{matrix} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ m, -m, \beta+\lambda, \beta-\lambda \end{matrix} \right. \right) dv \\ &= e^{-i \arg \zeta} \int_0^{\infty} G_{1,2}^{2,1} \left( e^{-i \arg \zeta} x \left| \begin{matrix} \frac{1}{2}+\kappa-\beta \\ \lambda-\beta, -\lambda-\beta \end{matrix} \right. \right) G_{2,4}^{2,1} \left( \zeta e^{-i \arg \zeta} x \left| \begin{matrix} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ m, -m, \beta+\lambda, \beta-\lambda \end{matrix} \right. \right) dx \\ &= G_{4,5}^{3,3} \left( \zeta \left| \begin{matrix} \beta-\lambda, \beta+\lambda, \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ \beta-\kappa-\frac{1}{2}, m, -m, \beta+\lambda, \beta-\lambda \end{matrix} \right. \right) = G_{1,2}^{2,1} \left( \zeta \left| \begin{matrix} \frac{1}{2}+k \\ m, -m \end{matrix} \right. \right) \\ &= \zeta^{-\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma(\tfrac{1}{2}-k+m) \Gamma(\tfrac{1}{2}-k-m) W_{k,m}(\zeta) \end{aligned}$$

wegen (60), womit der Beweis von Satz 3 erbracht ist.

Die Sätze 4, 5, 7, 8, 10 und 11 können auf analoge Weise aus Satz 1 abgeleitet werden.

Die Sätze 6, 9 und 12 hingegen sind Spezialfälle von Satz 2. Wegen (53) gilt nämlich

$$v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} M_{\kappa, \lambda}(v) = v^{\lambda-\beta} {}_1F_1\left(\frac{1}{2}-\kappa+\lambda; 1+2\lambda; v\right);$$

aus (49), (73) und (60) ergibt sich somit (ich setze  $v = e^{i\psi} x$ )

$$\begin{aligned} & \frac{-\kappa+\lambda}{1+2\lambda} \int_{-\infty e^{i\psi}}^{(0+)} v^{-\beta-\frac{1}{2}} e^{\frac{1}{2}v} M_{\kappa, \lambda}(v) G_{2,4}^{4,1}\left(\zeta v \left| \begin{array}{c} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ \beta+\lambda, \beta-\lambda, m, -m \end{array} \right. \right) dv \\ & \frac{e^{i\psi} \Gamma(\frac{1}{2}-\kappa+\lambda)}{\Gamma(1+2\lambda)} \int_{-\infty}^{(0+)} (e^{i\psi} x)^{\lambda-\beta} {}_1F_1\left(\frac{1}{2}-\kappa+\lambda; 1+2\lambda; e^{i\psi} x\right) G_{2,4}^{4,1}\left(\zeta e^{i\psi} x \left| \begin{array}{c} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2} \\ \beta+\lambda, \beta-\lambda, m, -m \end{array} \right. \right) dx \\ & 2\pi i G_{4,5}^{5,1}\left(\zeta \left| \begin{array}{c} \frac{1}{2}+k, \beta-\kappa-\frac{1}{2}, \beta-\lambda, \beta+\lambda \\ \beta-\kappa-\frac{1}{2}, \beta+\lambda, \beta-\lambda, m, -m \end{array} \right. \right) = 2\pi i G_{1,2}^{2,1}\left(\zeta \left| \begin{array}{c} \frac{1}{2}+k \\ m, -m \end{array} \right. \right) \\ & 2\pi i \zeta^{-\frac{1}{2}} e^{\frac{1}{2}\zeta} \Gamma\left(\frac{1}{2}-k+m\right) \Gamma\left(\frac{1}{2}-k-m\right) W_{k,m}(\zeta), \end{aligned}$$

womit der Beweis von Satz 6 geliefert ist.

Der Beweis der Sätze 9 und 12 geht ganz analog.

§ 5. Ich gebe jetzt ein Verzeichnis von Funktionen, die Spezialfälle von  $G_{p,q}^{m,n}(w)$  sind. Ich benutze hierbei die üblichen Bezeichnungen aus der Theorie der BESSELSchen und verwandten Funktionen.

$H_\nu^{(1)}(w)$  und  $H_\nu^{(2)}(w)$  sind die beiden HANKELschen Funktionen,  $J_\nu(w)$  und  $Y_\nu(w)$  die BESSELSchen Funktionen erster und zweiter Gattung; die Funktionen  $I_\nu(w)$  und  $K_\nu(w)$  können durch

$$I_\nu(w) = e^{-\frac{1}{2}\nu\pi i} J_\nu(w e^{\frac{1}{2}\pi i}), \quad . \quad . \quad . \quad . \quad . \quad (77)$$

und

$$K_\nu(w) = \frac{1}{2}\pi i e^{\frac{1}{2}\nu\pi i} H_\nu^{(1)}(w e^{\frac{1}{2}\pi i}) = \frac{\pi}{2\sin\nu\pi} \{I_{-\nu}(w) - I_\nu(w)\} \quad . \quad (78)$$

definiert werden.

$S_{\mu,\nu}(w)$  bezeichnet die LOMMELsche Funktion<sup>24)</sup>;  $\mathbf{H}_\nu(w)$  und  $\mathbf{L}_\nu(w) = e^{-\frac{1}{2}(\nu+1)\pi i} \mathbf{H}_\nu(w e^{\frac{1}{2}\pi i})$  sind die beiden STRUVESchen Funktionen<sup>25)</sup>.

Ferner setze ich noch

$$U_{\nu,\mu}(w) = \frac{1}{2i} \{e^{\frac{1}{2}(\nu-\mu)\pi i} H_\nu^{(1)}(w) H_\mu^{(2)}(w) - e^{\frac{1}{2}(\mu-\nu)\pi i} H_\mu^{(1)}(w) H_\nu^{(2)}(w)\}$$

und

$$V_{\nu,\mu}(w) = \frac{1}{2} \{e^{\frac{1}{2}(\nu-\mu)\pi i} H_\nu^{(1)}(w) H_\mu^{(2)}(w) + e^{\frac{1}{2}(\mu-\nu)\pi i} H_\mu^{(1)}(w) H_\nu^{(2)}(w)\}. \quad (79)$$

<sup>24)</sup> WATSON, [26], 347—349.

<sup>25)</sup> WATSON, [26], 328—329.

Unter Annahme dieser Bezeichnungen hat man <sup>26)</sup>

$$J_{\nu}(w) = G_{0,2}^{1,0} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} \nu, -\frac{1}{2} \nu \right), \quad . \quad . \quad . \quad . \quad . \quad (80)$$

$$K_{\nu}(w) = \frac{1}{2} G_{0,2}^{2,0} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} \nu, -\frac{1}{2} \nu \right), \quad . \quad . \quad . \quad . \quad . \quad (81)$$

$$Y_{\nu}(w) = (-1)^h G_{1,3}^{2,0} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} \nu, -\frac{1}{2} \nu, -\frac{1}{2} \nu - h - \frac{1}{2} \right) \quad (h=0, \pm 1, \pm 2, \dots), \quad (82)$$

$$H_{\nu}(w) = G_{1,3}^{1,1} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} + \frac{1}{2} \nu, \frac{1}{2} + \frac{1}{2} \nu, -\frac{1}{2} \nu, \frac{1}{2} \nu \right), \quad . \quad . \quad . \quad . \quad . \quad (83)$$

$$I_{\nu}(w) - L_{\nu}(w) = \frac{1}{\pi} G_{1,3}^{2,1} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} + \frac{1}{2} \nu, \frac{1}{2} + \frac{1}{2} \nu, -\frac{1}{2} \nu, \frac{1}{2} \nu \right), \quad . \quad . \quad . \quad (84)$$

$$I_{-\nu}(w) - L_{\nu}(w) = \frac{\cos \nu \pi}{\pi} G_{1,3}^{2,1} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} + \frac{1}{2} \nu, \frac{1}{2} + \frac{1}{2} \nu, -\frac{1}{2} \nu, \frac{1}{2} \nu \right), \quad . \quad (85)$$

$$H_{\nu}(w) - Y_{\nu}(w) = \frac{\cos \nu \pi}{\pi^2} G_{1,3}^{3,1} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} + \frac{1}{2} \nu, \frac{1}{2} + \frac{1}{2} \nu, -\frac{1}{2} \nu, \frac{1}{2} \nu \right), \quad . \quad (86)$$

$$S_{\mu, \nu}(w) = \frac{2^{\mu-1}}{\Gamma(\frac{1}{2} - \frac{1}{2} \mu + \frac{1}{2} \nu) \Gamma(\frac{1}{2} - \frac{1}{2} \mu - \frac{1}{2} \nu)} G_{1,3}^{3,1} \left( \frac{1}{4} w^2 \middle| \frac{1}{2} + \frac{1}{2} \mu, \frac{1}{2} + \frac{1}{2} \mu, -\frac{1}{2} \nu, \frac{1}{2} \nu \right), \quad (87)$$

$$J_{\nu}^2(w) = \frac{1}{\sqrt{\pi}} G_{1,3}^{1,1} \left( w^2 \middle| \frac{1}{2}, \nu, -\nu, 0 \right), \quad . \quad . \quad . \quad . \quad . \quad (88)$$

$$J_{\nu}(w) J_{-\nu}(w) = \frac{1}{\sqrt{\pi}} G_{1,3}^{1,1} \left( w^2 \middle| \frac{1}{2}, 0, \nu, -\nu \right), \quad . \quad . \quad . \quad . \quad . \quad (89)$$

$$J_{\nu}(w) Y_{\nu}(w) = -\frac{1}{\sqrt{\pi}} G_{1,3}^{2,0} \left( w^2 \middle| \frac{1}{2}, 0, \nu, -\nu \right), \quad . \quad . \quad . \quad . \quad . \quad (90)$$

$$J_{-\nu}^2(w) - J_{\nu}^2(w) = \frac{2 \sin \nu \pi}{\sqrt{\pi}} G_{1,3}^{2,0} \left( w^2 \middle| \frac{1}{2}, \nu, -\nu, 0 \right), \quad . \quad . \quad . \quad . \quad . \quad (91)$$

$$K_{\nu}^2(w) = \frac{1}{2} \sqrt{\pi} G_{1,3}^{3,0} \left( w^2 \middle| \frac{1}{2}, 0, \nu, -\nu \right), \quad . \quad . \quad . \quad . \quad . \quad (92)$$

<sup>26)</sup> Siehe [18], 12—17; [13], 397—398; [14], 138; [16], 206—207.

Für (107) vergl. man [15], 885, Formel (111).

(105) folgt aus (56) und (1) wegen

$$\begin{aligned} {}_1F_1(a; b; z) &= {}_2F_3\left(\frac{1}{2}a, \frac{1}{2}a + \frac{1}{2}; \frac{1}{2}b, \frac{1}{2}b + \frac{1}{2}, \frac{1}{2}; \frac{1}{4}z^2\right) \\ &+ \frac{az}{b} {}_2F_3\left(\frac{1}{2}a + \frac{1}{2}, \frac{1}{2}a + 1; \frac{1}{2}b + \frac{1}{2}, \frac{1}{2}b + 1, \frac{3}{2}; \frac{1}{4}z^2\right). \end{aligned}$$

$$I_{\nu}(w) K_{\nu}(w) = \frac{1}{2\sqrt{\pi}} G_{1,3}^{2,1} \left( w^2 \left| \begin{matrix} \frac{1}{2} \\ 0, \nu, -\nu \end{matrix} \right. \right), \quad (93)$$

$$I_{-\nu}^2(w) - I_{\nu}^2(w) = \frac{\sin 2\nu\pi}{\pi^{\frac{3}{2}}} G_{1,3}^{2,1} \left( w^2 \left| \begin{matrix} \frac{1}{2} \\ \nu, -\nu, 0 \end{matrix} \right. \right), \quad (94)$$

$$H_{\nu}^{(1)}(w) H_{\nu}^{(2)}(w) = \frac{2 \cos \nu\pi}{\pi^{\frac{3}{2}}} G_{1,3}^{3,1} \left( w^2 \left| \begin{matrix} \frac{1}{2} \\ 0, \nu, -\nu \end{matrix} \right. \right), \quad (95)$$

$$J_{\nu}(w) J_{\mu}(w) = \frac{1}{\sqrt{\pi}} G_{2,4}^{1,2} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{matrix} \right. \right), \quad (96)$$

$$\left. \begin{aligned} J_{\nu}(w) J_{\mu}(w) + J_{-\nu}(w) J_{-\mu}(w) &= \frac{2 \cos (\frac{1}{2}\nu + \frac{1}{2}\mu) \pi}{\sqrt{\pi}} \\ &\times G_{2,4}^{2,1} \left( w^2 \left| \begin{matrix} \frac{1}{2}, 0 \\ \frac{1}{2}\nu + \frac{1}{2}\mu, -\frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu \end{matrix} \right. \right), \end{aligned} \right\} \quad (97)$$

$$\left. \begin{aligned} J_{\nu}(w) J_{\mu}(w) - J_{-\nu}(w) J_{-\mu}(w) &= -\frac{2 \sin (\frac{1}{2}\nu + \frac{1}{2}\mu) \pi}{\sqrt{\pi}} \\ &\times G_{2,4}^{2,1} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, -\frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu \end{matrix} \right. \right), \end{aligned} \right\} \quad (98)$$

$$\left. \begin{aligned} H_{\nu}^{(1)}(w) H_{\mu}^{(1)}(w) - H_{\nu}^{(2)}(w) H_{\mu}^{(2)}(w) \\ = \frac{4}{\sqrt{\pi} i} G_{2,4}^{3,0} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{matrix} \right. \right), \end{aligned} \right\} \quad (99)$$

$$K_{\nu}(w) K_{\mu}(w) = \frac{1}{2\sqrt{\pi}} G_{2,4}^{4,0} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{matrix} \right. \right), \quad (100)$$

$$I_{\nu}(w) K_{\mu}(w) = \frac{1}{2\sqrt{\pi}} G_{2,4}^{2,2} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{matrix} \right. \right), \quad (101)$$

$$\left. \begin{aligned} I_{-\nu}(w) I_{-\mu}(w) - I_{\nu}(w) I_{\mu}(w) &= \frac{\sin (\nu + \mu) \pi}{\pi^{\frac{3}{2}}} \\ &\times G_{2,4}^{2,2} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, -\frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu \end{matrix} \right. \right), \end{aligned} \right\} \quad (102)$$

$$\left. \begin{aligned} U_{\nu, \mu}(w) &= \frac{2 \sin (\frac{1}{2}\nu + \frac{1}{2}\mu) \pi \sin (\frac{1}{2}\nu - \frac{1}{2}\mu) \pi}{\pi^{\frac{3}{2}}} \\ &\times G_{2,4}^{4,1} \left( w^2 \left| \begin{matrix} 0, \frac{1}{2} \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{matrix} \right. \right), \end{aligned} \right\} \quad (103)$$



$$\left. \begin{aligned} V_{\nu, \mu}(w) &= \frac{2 \cos(\frac{1}{2}\nu + \frac{1}{2}\mu) \pi \cos(\frac{1}{2}\nu - \frac{1}{2}\mu) \pi}{\pi^{\frac{1}{2}}} \\ &\times G_{2,4}^{4,1} \left( w^2 \left| \begin{array}{c} \frac{1}{2}, 0 \\ \frac{1}{2}\nu + \frac{1}{2}\mu, \frac{1}{2}\nu - \frac{1}{2}\mu, \frac{1}{2}\mu - \frac{1}{2}\nu, -\frac{1}{2}\nu - \frac{1}{2}\mu \end{array} \right. \right), \end{aligned} \right\} \quad (104)$$

$$e^{-w} M_{k,m}(2w) = \frac{2^k w^{\frac{1}{2}} \Gamma(1+2m)}{\sqrt{\pi} \Gamma(\frac{1}{2}+k+m)} G_{2,4}^{2,2} \left( w^2 \left| \begin{array}{c} \frac{3}{4} - \frac{1}{2}k, \frac{1}{4} - \frac{1}{2}k \\ \frac{1}{2} + \frac{1}{2}m, \frac{1}{2}m, \frac{1}{2} - \frac{1}{2}m, -\frac{1}{2}m \end{array} \right. \right), \quad (105)$$

$$e^{-w} W_{k,m}(2w) = \frac{2^k w^{\frac{1}{2}}}{\sqrt{\pi}} G_{2,4}^{4,0} \left( w^2 \left| \begin{array}{c} \frac{3}{4} - \frac{1}{2}k, \frac{1}{4} - \frac{1}{2}k \\ \frac{1}{2} + \frac{1}{2}m, \frac{1}{2} - \frac{1}{2}m, \frac{1}{2}m, -\frac{1}{2}m \end{array} \right. \right), \quad (106)$$

$$M_{k,m}(iw) M_{k,m}(-iw) = \frac{\sqrt{\pi} w \Gamma^2(1+2m)}{\Gamma(\frac{1}{2}+k+m) \Gamma(\frac{1}{2}-k+m)} G_{2,4}^{1,2} \left( \frac{1}{4} w^2 \left| \begin{array}{c} \frac{1}{2}+k, \frac{1}{2}-k \\ m, -m, \frac{1}{2}, 0 \end{array} \right. \right), \quad (107)$$

$$M_{k,m}(w) W_{-k,m}(w) = \frac{w \Gamma(1+2m)}{2 \sqrt{\pi} \Gamma(\frac{1}{2}+k+m)} G_{2,4}^{3,1} \left( \frac{1}{4} w^2 \left| \begin{array}{c} \frac{1}{2}-k, \frac{1}{2}+k \\ m, \frac{1}{2}, 0, -m \end{array} \right. \right), \quad (108)$$

$$W_{k,m}(iw) W_{k,m}(-iw) = \frac{w}{2 \sqrt{\pi} \Gamma(\frac{1}{2}-k+m) \Gamma(\frac{1}{2}-k-m)} G_{2,4}^{4,1} \left( \frac{1}{4} w^2 \left| \begin{array}{c} \frac{1}{2}+k, \frac{1}{2}-k \\ m, -m, \frac{1}{2}, 0 \end{array} \right. \right), \quad (109)$$

$$W_{k,m}(w) W_{-k,m}(w) = \frac{w}{2 \sqrt{\pi}} G_{2,4}^{4,0} \left( \frac{1}{4} w^2 \left| \begin{array}{c} \frac{1}{2}+k, \frac{1}{2}-k \\ m, -m, \frac{1}{2}, 0 \end{array} \right. \right). \quad (110)$$


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**Mathematics.** — *Théorème sur l'itération d'une représentation conforme.*  
(Deuxième communication). Par Prof. J. WOLFF. (Communicated  
by Prof. J. G. VAN DER CORPUT.)

(Communicated at the meeting of January 25, 1941.)

Soit  $D_1$  un domaine intérieur au demi-plan  $D$  ( $\text{R}z > 0$ ) et dont la frontière  $f$  est une courbe de Jordan intérieure à  $D$ , s'étendant à l'infini dans les deux directions  $\arg z \rightarrow -\frac{\pi}{2}$  et  $\arg z \rightarrow \frac{\pi}{2}$ . Représentons  $D$  conformément sur  $D_1$  tel que  $z_1(1) = 1$ ,  $z_1(\infty) = \infty$ . Soit  $z_1(z)$  la fonction représentatrice,  $\lambda$  sa dérivée angulaire à l'infini.

La première communication (Proceedings Vol. XLIII, No. 8, 1940, p. 1016) contient le théorème:

A. Si l'ensemble  $N$  commun aux domaines itérés  $D_1, D_2, \dots$  contient un domaine  $\Delta$  contenant un point  $a$  et son image  $a_1 = z_1(a)$ , alors  $\lambda > 0$ .

Cependant la démonstration s'est appuyée sur une condition accessoire imposée à la frontière  $f$  de  $D_1$ . Dans la présente communication nous montrerons d'abord que le théorème A n'exige aucune autre hypothèse par rapport à la frontière  $f$  que celle faite ci-dessus.

Puis nous démontrerons le théorème:

B. Si  $N$  contient un domaine  $\Delta$  dont  $z=1$  et  $z=\infty$  sont des points-frontières, alors  $\lambda > 0$  et  $N$  contient un domaine  $G$  dont l'ouverture à l'infini égale  $\pi$ .

*Démonstration de A.* En appliquant la même méthode que dans la première communication (p. 1016 et 1017) on conclut que, sur tout ensemble fermé et borné dans  $\Delta$ ,

$$0 < c < \left| \frac{z_{-(n+1)}}{z_{-n}} \right| < C < \infty, \quad n=0, 1, 2, \dots, c \text{ et } C \text{ fixes.} \quad (1)$$

Traçons dans  $\Delta$  un polygone  $\Gamma$  d'extrémités  $a_1$  et  $a$ . Puisque  $z_{-n} \rightarrow \infty$  pour  $n \rightarrow \infty$ , la réunion des antécédents  $\Gamma_{-n}$  de  $\Gamma$  est une courbe  $\gamma$  s'étendant à l'infini.

1. Si  $\gamma$  contient une suite de points  $\rightarrow \infty$ , situés dans un angle  $|\arg| < \frac{\pi}{2} - \varepsilon$ ,  $\varepsilon$  positif et fixe, alors (1) montre que  $\lambda \equiv C^{-1} > 0$ .

2. Dans le cas contraire on a sur  $\gamma$

$$\lim_{z \rightarrow \infty} \arg z = \frac{\pi}{2} \text{ ou } -\frac{\pi}{2}.$$

Il suffit de considérer le cas  $\arg z \rightarrow \frac{\pi}{2}$ . Alors sur  $\Gamma$

$$\lim_{n \rightarrow \infty} \arg z_{-n} = \frac{\pi}{2} \quad \dots \quad (2)$$

La suite des fonctions  $\arg z_{-n}$ , harmoniques dans  $\Delta$ , étant uniformément bornée, elle est normale dans  $\Delta$ . On en conclut que (2) est valable dans  $\Delta$ . En effet, admettons pour un instant que  $\Delta$  contienne un point  $\beta$  tel que, pour une suite  $(p)$  d'entiers  $p \rightarrow \infty$ ,  $\arg \beta_{-p} < \frac{\pi}{2} - \varepsilon$ ,  $\varepsilon > 0$  et fixe. De la suite  $(p)$  on pourrait extraire une suite  $q \rightarrow \infty$  telle que dans  $\Delta$   $\arg z_{-q}$  tendrait vers une fonction harmonique  $\varphi(z)$ . Alors  $\varphi(\beta) \equiv \frac{\pi}{2} - \varepsilon$  et sur  $\Gamma$   $\varphi(z) = \frac{\pi}{2}$ . Or le maximum de  $\varphi(z)$  serait  $\frac{\pi}{2}$ , donc  $\varphi(z) = \frac{\pi}{2}$  en tout point de  $\Delta$ , en contradiction avec  $\varphi(\beta) < \frac{\pi}{2}$ .

Par conséquent (2) est valable dans  $\Delta$ . La méthode de la première communication conduit maintenant à

$$\lim_{n \rightarrow \infty} \log \left| \frac{z_{-n}}{z_{-(n+1)}} \right| = 0 \text{ dans } \Delta,$$

donc en vertu de  $\arg z_{-n} \rightarrow \frac{\pi}{2}$ :

$$\lim_{n \rightarrow \infty} \frac{z_{-n}}{z_{-(n+1)}} = 1 \text{ dans } \Delta, \text{ d'où}$$

$$\lim_{z \rightarrow \infty} \frac{z_1(z)}{z} = 1 \text{ sur la courbe } \gamma.$$

La fonction  $\frac{z_1(z)}{z}$ , holomorphe dans  $D$ , ne prend pas les valeurs négatives, donc elle ne peut pas tendre vers des limites différentes sur deux courbes dans  $D$  tendant vers l'infini. Or elle tend vers  $\lambda$  sur l'axe réel, donc  $\lambda = 1$ , en contradiction avec  $z_1(1) = 1$ . Par suite: le cas 2 ne se produit pas.

*Démonstration de B.* Considérons le domaine  $\Delta_1$  lieu des itérés  $z_1(z)$  des points  $z$  de  $\Delta$ . Si  $\Delta$  et  $\Delta_1$  ont un point commun  $\alpha_1$ , alors le domaine  $\Delta + \Delta_1$  satisfait aux prémisses du théorème A, donc  $\lambda > 0$ .

Dans le cas contraire remarquons que  $z = 1$  et  $z = \infty$  sont des points-frontières de  $\Delta_1$  comme de  $\Delta$ . Soit  $n$  un entier positif. Le domaine  $D_n$  contient  $\Delta$  et  $\Delta_1$ . Puisque  $D_n$  est simplement connexe, il contient le plus petit domaine simplement connexe  $\overline{\Delta}$  contenant  $\Delta$  et  $\Delta_1$ . Cela étant pour  $n = 1, 2, \dots$ ,  $\overline{\Delta}$  appartient à  $N$ . De plus  $\overline{\Delta}$  satisfait aux prémisses du théorème A, donc  $\lambda > 0$ .

Or dans une note présentée à la fin d'Avril 1940 aux *Comptes Rendus* de l'Ac. des Sc., Paris, j'ai montré que

C. Si  $\lambda > 0$ , alors  $N$  contient un domaine  $G$  dont l'ouverture à l'infini égale  $\pi$ .

Donc le théorème B est démontré. Une conséquence des résultats obtenus est l'énoncé suivant:

D. La condition nécessaire et suffisante pour que  $N$  contienne un domaine  $G$  d'ouverture  $\pi$  à l'infini est que  $\lambda > 0$ .

En effet, la condition est nécessaire: grâce à l'hypothèse faite sur la frontière  $f$  de  $D_1$ , l'argument de  $z_1/z$  tend vers zéro pour  $z \rightarrow \infty$ , donc si  $a$  est réel et suffisamment grand, les points  $a$  et  $a_1 = z_1(a)$  sont dans  $G$ ;  $\lambda > 0$  en vertu du théorème A. Le théorème C montre que la condition est suffisante.

De plus le raisonnement dans la note citée conduit à:

E. Dans  $G$  l'argument de  $z_{-n}$  tend pour  $n$  infini vers une fonction harmonique prenant toutes les valeurs entre  $-\frac{\pi}{2}$  et  $\frac{\pi}{2}$ .

Enfin signalons que

F. Si  $\lambda = 0$  et si  $N$  contient un domaine  $\Delta$ , alors  $N$  contient l'ensemble infini des domaines  $\Delta_n$  et  $\Delta_{-n}$ ,  $n = 1, 2, \dots$ , extérieurs l'un à l'autre.

Il est facile de réaliser ce cas au moyen d'une transformation de KOENIGS.



**Medicine.** — *A contribution to our knowledge about the conduction of stimuli in the auditory nerve.* By J. BIJTEL. (Communicated by Prof. A. DE KLEYN.)

(Communicated at the meeting of January 25, 1941.)

The problem of the character of the conduction of acoustic stimuli to the central nervous system by the auditory nerve is of important significance for our knowledge of the physiology of hearing. We can formulate the problem as follows: a conduction, having the character of physico-chemical undulations, more or less of like form as the mechanical undulations of the sound, should indicate an analysis of the sound in the central nervous system, whereas a similar conduction in the various nerve fibres, independent from the mechanical undulations of the sound, could be taken as evidence for peripheral analysis. Central or peripheral analysis is the alternative in modern conceptions. Several pro- and con-arguments can be advanced for both conceptions. However, we confine to indicate but a few data.

The electrical responses of the cochlea, as far as they are thought to be action-currents of the cochlear nerve, are, to a certain extent, of like form as the mechanical waves of the stimulating sound. This phenomenon makes a central analysis probable. The structure of the cochlear organ suggests the improbability of an exact peripheral analysis in physical sense. On the other hand some symptoms, observed in harmonics, formed in the ear itself, add support to the idea of peripheral analysis. Summarizing, it is evident that the problem is not completely solved and that further experiments on this subjects are necessary, while those experiments, about which no irrefutable conception exists, must be repeated.

If the conduction in the nerve is depending on oscillations of a physico-chemical character, of like form as the mechanical oscillations of the sound, it is clear that beats must develop in the central nervous system when two sounds, differing a few periods in pitch, are presented to the ears of one person, so that to each ear one sound is presented. We know that the stimuli from both ears meet in the central nervous system. If two sounds of the same pitch, both below the threshold of hearing, are presented to the ears of the same person (to each ear one sound) they can be summarized to an audible intensity. These considerations made us to perform a simple investigation by which it became evident that in binaural hearing beats are not perceived. We presented the frequency 512 or 1024 to one ear and introduced a tone, differing from this 1 or 2 cycles per second to the other ear. Here must be pointed to the fact that it is absolutely necessary that

one ear perceives only one of the two tones. As soon as these sounds interfere physically in the air or in the skull, beats are perceived. The experiments were arranged in the following way. Before the external meatus of one ear a weak sounding tuning fork c 2 or c 3 was placed. To the other ear a tone of one or two cycles per second difference was introduced into the external meatus through a rubber tube. This was so performed that overhearing by the first ear was impossible. The other end of the rubber tube was connected with a leaden tube which caught the tone from a loudspeaker in another room. Under these circumstances no beats are heard. If, however, the occlusion of the rubber tube is insufficient, beats are heard if a sufficient intensity of the tuning fork tone exists. These beats are also perceived when the butt of the tuning fork is placed on the skull, introducing its tone into the ear, in which the rubber tube is placed, or on the rubber tube itself.

In *Physical Reviews*, vol. 26, 1925, we found a communication of LANE on Binaural Beats. LANE makes difference between objective and subjective beats. According to LANE the subjective beats should have their origin in the central nervous system, and are only perceived in frequencies up to 700 or 800 cycles per second. He thinks the localisation-effects of these beats an indication for the fact that they are closely connected to the ability to perceive phase differences. According to him the localisation of the sound in space, in frequencies up to 700—800 cycles per second, should also be due to perception of the differences in phase.

As in our experiments, 11 persons, without exception, did not perceive beats in binaural hearing, we think that in LANE's experiments the tones, presented to the ears, were not sufficiently separated. It is impossible for us to explain by other facts the difference between LANE's and our experiments.

From our observations we draw the conclusion that conduction through the auditory nerve cannot have the character of oscillations of like form as the mechanical oscillations of the stimulating tone. However, to our opinion, peripheral analysis is not yet proved in this way.

**Medicine.** — *L'effet prismatique dans les cas-MÉNIÈRE.*

Par G. P. UTERMÖHLEN. (Communicated by Prof. A. DE KLEIN.)

(Communicated at the meeting of January 25, 1941.)

En l'année 1861 M. PROSPER MÉNIÈRE fit part à l'Académie Impériale de Paris d'une maladie de l'appareil auditif caractérisée par des symptômes situés dans le domaine de troubles de l'ouïe et de l'équilibre. Sa conclusion dans le Bulletin de l'Académie du 8 janvier 1861 était la suivante:

„1. Un appareil auditif, jusque-là parfaitement sain, peut devenir tout à coup le siège de troubles fonctionnels, consistants en bruits de nature variable, continus ou intermittents et ces bruits s'accompagnent bientôt d'une diminution plus ou moins grande de l'audition.

2. Ces troubles, ayant leur siège dans l'appareil auditif, peuvent donner lieu à des accidents réputés cérébraux, tels que vertige, étourdissements, marche incertaine, tournoissements et chute, et de plus ils sont accompagnés de nausées, de vomissements et d'un état syncopal.

3. Ces accidents, qui ont la forme intermittente, ne tardent pas à être suivis de surdité de plus en plus grave et souvent l'ouïe est subitement et complètement abolie.

4. Tout porte à croire que la lésion matérielle, qui est cause de ces troubles fonctionnels, réside dans les canaux semi-circulaires.

A l'autopsie de sa malade il fut constaté une hémorragie dans les canaux semicirculaires.

La description de ce syndrome classique, auquel le nom de MÉNIÈRE est lié avec raison, n'a rien perdu de sa valeur depuis la première communication de celui-ci. Toutefois il s'est révélé plus tard que dans la plupart des cas la cause des symptômes que MÉNIÈRE décrivit si succinctement et correctement ne peut pas seulement être attribuée à des hémorragies dans les canaux semicirculaires mais qu'une grande variété d'affections de l'organe périphérique, du N. octavus et même du domaine central du N. octavus peuvent causer les mêmes symptômes. Il est même probable que chez le groupe le plus considérable des malades soi-disant souffrants de „MÉNIÈRE” chez qui les attaques de vertige et les troubles de l'ouïe alternent avec des périodes où le malade se trouve plus ou moins en bonne santé, ce ne sont pas des causes organiques qui jouent le rôle principal, mais des troubles purement vasomoteurs qui causent les symptômes.

Je suis pour cela d'avis qu'il vaut mieux réserver le concept „maladie de MÉNIÈRE” à l'affection originelle décrite par MÉNIÈRE et de classer tous les autres cas sous le nom de „syndrome de MÉNIÈRE”.

Dans la communication suivante il ne sera question que de malades souffrant d'attaques intermittentes de vertige et de troubles de l'ouïe chez qui, hormis une surdité plus ou moins grave et un équilibre labile des organes de l'équilibre, on ne rencontre pas de sérieuses anomalies dans le domaine de l'otologie ou de la neurologie (comme p.e. la labyrinthite, traumatismes du labyrinthe, névrite acoustique, tumeur acoustique etc.).

La période, située entre les attaques proprement dites est souvent caractérisée chez les malades souffrant des symptômes susnommés, par l'apparition de légers vertiges, de nausées, d'une démarche incertaine, lorsque lesdits malades changent de position, et de troubles qui ont lieu lorsque le malade change la direction de son regard.

A un moment donné il se peut que, par des circonstances secondaires, comme p.e. après un éternuement qui augmente la tension dans l'oreille moyenne, par toute sorte d'irritations, voire même d'après une certaine communication, par l'affaissement de la voûte du pied, une véritable attaque se produise avec des sensations très désagréables pour le malade comme p.e. les mains et les pieds froids, une forte sueur, une désorientation absolue, vomissements et diarrhée, le sentiment d'être absolument impuissant, tournoiements et nystagmus. Le nystagmus peut être horizontal, rotatoire et une combinaison des deux. Dans le cas d'attaques sérieuses de vertige qui sont généralement accompagnées de nystagmus d'une fréquence très élevée, il arrive que le malade tombe subitement sans perdre conscience. Il n'y a pas longtemps un malade souffrant de pareils symptômes fut blessé mortellement.

Il va de soi que les malades qui souffrent d'attaques semblables doivent être considérés comme absolument invalides.

Au cours des années on a appliqué différentes méthodes de guérison. Des médicaments comme la chinine, les bromures, hypnotiques, véronal, bellergal, atropine, morphine (ce dernier contre les vomissements souvent violents et prolongés), monotrén (qui renferme de la chinine et de la papavérine) étaient administrés. Des régimes spéciaux ou des cures au sanatorium, des cures de sommeil et de repos étaient prescrits. Mais tout ceci n'avait souvent que des résultats temporaires ou absolument négatifs.

Une aggravation de la surdité fait souvent diminuer la fréquence des attaques. C'est sur ce fait qu'est basée la thérapie de CHARCOT qui essayait de provoquer une surdité par une grande dose de chinine.

Les périodes entre les crises se caractérisaient le plus souvent chez les malades traités par moi-même par la peur d'une répétition des crises et c'est cette peur aussi qui rendait les malades plus ou moins invalides, comme ils n'osaient se mouvoir dans les rues sans être soutenus et guidés par quelqu'un.

Chez l'un des malades observés, l'attention de l'auteur de la présente communication, qui pouvait suivre très exactement tout le cours de la crise de vertige, fut attirée par une légère exophtalmie de l'oeil, du côté homolatéral de l'oreille qui était atteinte d'une catarrhe chronique de l'oreille moyenne.



Durant l'attaque il constata un nystagmus d'une très grande fréquence qui était dirigé horizontalement-rotatoirement vers le côté de l'oreille malade et qui se modifia ensuite vers la fin de l'attaque en un nystagmus à direction opposée. En réfléchissant sur cet état il se demanda si une action optique ne pourrait d'une manière ou de l'autre exercer une influence. Peut-être le réflexe optique de la mise au point? *En effet ceci parut être le cas.* Car en tenant devant l'oeil du côté de l'oreille malade un prisme, dont la base était posée dans la direction du nez, il effectua *immédiatement* un soulagement. Par contre, le placement d'un prisme dont la base fut placée dans la direction de la tempe provoqua une aggravation des sensations très désagréables. Par ce fait il devint peu à peu évident que chez ce malade l'effort de la convergence, fait pour voir au loin et plus encore pour la vue à courte distance, jouait un grand rôle dans la production des attaques de vertige et dans la persistance des troubles de l'équilibre moins importants, pendant les intervalles entre les attaques. Plus tard on trouva chez différents malades que l'effort de la convergence est encore plus grand pour beaucoup d'hypermétropes qui montrent souvent de l'ésophorie et que pour des malades emmétropes souffrant de vertiges, ce facteur peut être également d'une grande signification. Depuis lors on appliqua chez une vingtaine de malades souffrant du syndrome de MÉNIÈRE, dont une partie fut examinée en collaboration avec le Professeur Dr. A. DE KLEYN dans sa clinique, l'effet prismatique avec un même résultat favorable.

Lorsqu'on place un verre prismatique — l'arête dans la direction verticale — p.e. devant l'oeil droit, il se produit une image double lorsque le patient regarde binoculairement un objet à fixer. Il dépend de la position du prisme dans quelle direction l'oeil se tournera pour former une image simple en confusionnant l'image double. Lorsque l'apex est dirigé du côté du nez, l'oeil se tourne également de ce côté, c.à.d. en dedans, et de la même manière l'oeil suit l'apex lorsque celui-ci est placé dans la direction de la tempe. En plaçant le prisme avec sa base vers le côté du nez, le *musculus rectus internus oculi* se détend et contracte le *m. rectus externus*. Dans les cas non-complicés du syndrome de MÉNIÈRE, la détente des *mm. recti interni* a une action favorable qui se fait sentir par le malade comme extrêmement soulageant. Ainsi chez mes malades le placement du prisme n'avait un bon résultat que dans le cas où la base en était placée dans la direction du nez.

Pour un malade qui a de l'ésophorie il est évident que l'effet du traitement susnommé doit être indiscutable, par le fait qu'il existe déjà — surtout si la réfraction de l'oeil est hypermétrope — une action prédominante des *mm. recti interni*.

Or, que se passe-t-il dans le cas d'exophorie?

Ici, lorsque le malade regarde au loin, il se produit une divergence des axes optiques, qui, sous les mêmes conditions, se dirigent parallèlement par une position normale des yeux.

Lorsqu'on veut porter l'image de l'oeil divergent sur la fovea centralis retinae on peut le faire de deux manières.

1. L'oeil peut, par l'action du m. rectus internus éliminer la divergence, ce qui s'effectue non sans peine. Le malade, ayant de l'exophorie, est, sans le savoir, continuellement occupé à faire ceci. On s'en aperçoit très clairement lorsqu'on examine le malade au moyen d'un verre MADDOX.

2. En supposant que l'oeil ne bouge pas, on peut obtenir l'image sur la fovea centralis retinae en plaçant un prisme très faible devant l'oeil divergent. En faisant ceci on élimine le besoin de confusionner, vu que la formation de l'image s'est effectuée. L'effort que cela coûterait devient donc superflu.

Chez un malade souffrant du syndrome de MÉNIÈRE qui a de l'exophorie la correction optiquement appliquée de la réduction d'une image double en une image simple par un prisme dont la base est dirigée du côté du nez, produit une sensation agréable.

Par cela on obtient une diminution du facteur de la convergence qui produit une crise. Si cette diminution de la valeur du facteur de convergence ne serait pas encore obtenue, l'on pourrait également placer un prisme devant l'autre oeil, par quoi il est probable que le but sera atteint.

Un exophorique a sans prisme — le regard étant au repos — pour ainsi dire un oeil placé normalement lequel, s'adaptant à un prisme dont l'apex est dirigé vers la tempe, a par ce fait une tendance à dévier du côté de la tempe.

Lorsqu'on retourne le prisme mentalement, il redevient pour ainsi dire un oeil dans sa position normale.

Par ce fait s'explique le paradoxe situé dans la règle générale que la base doit être placée dans la direction du nez et que ceci doit être le cas tant pour l'ésophorie que pour l'exophorie.

Une fois l'oeil exophorique corrigé, la convergence à la mise au point à courte distance suit binoculairement son cours habituel.

Cette application importante chez les malades souffrant du syndrome de MÉNIÈRE, indique que *l'énergie de convergence* peut influencer un équilibre dans les actions des noyaux des centres vestibulaires, lequel chez ces malades est d'un caractère labile, de telle façon qu'une rupture de cet équilibre avec les conséquences inhérentes (action sur le centre vague etc.) ne s'effectue pas.

Nous pouvons dire maintenant avec une certitude assez grande que l'effet d'un verre prismatique faible d'un angle réfractaire d'un demi, d'un, d'un et demi, de deux, au plus de deux degrés et demi, *peut protéger* les malades dans les périodes intermittentes entre les crises et de façon préventive à l'égard de la crise même.

La prescription d'un verre prismatique doit se faire préalablement de façon plus ou moins empirique. Il s'est démontré que souvent il suffit qu'on applique un prisme d'un demi degré à l'un des yeux (p.e. l'oeil exophorique c.q.) et un verre ordinaire à l'autre.

S'il se trouve que seulement un prisme de deux degrés donne de bons résultats, l'on peut placer devant chaque oeil un prisme d'un degré, à condition que la base en soit toujours dirigée du côté du nez.

D'après mon expérience il faut, dans le cas où les yeux du malade ont une déviation réfractaire, placer le prisme le plus fort devant l'oeil avec la déviation la plus grave. Pour un hypermétrope il est nécessaire de prendre un prisme d'un ou d'un et demi degré. Dans un cas  $\Delta 3^\circ$  sur l'oeil droit et  $\Delta 1^\circ$  sur l'oeil gauche donna le résultat désiré.

Le fait le plus remarquable qui s'est fait jour, consiste en ce que nos malades chez qui se manifesta le syndrome de MÉNIÈRE ressentirent *immédiatement* l'action favorable du prisme et se métamorphosèrent en un moment d'invalides en hommes sains, capables de faire tous les mouvements voulus.

L'on peut se servir le mieux de verres *plans*, biconvexes ou biconcaves, mais pas de verres convexes-concaves, à cause de la grande incommodité des reflexes de lumière qui s'y produisent. Les malades doivent ou se servir continuellement de leurs lunettes ou les avoir à portée de la main. Il faudra qu'ils se gardent de se mouvoir dans l'obscurité pour éviter l'effort de pouvoir discerner les objets autour d'eux.

### Conclusion.

Lorsqu'on indique la valeur stimulante totale qui est nécessaire pour l'effectuation du syndrome de MÉNIÈRE, donc pour provoquer une attaque, par le caractère T; la valeur des stimulants émis par le labyrinthe, respectivement par les deux labyrinthes, par le caractère L; l'énergie (le stimulant) nécessaire à la convergence par le caractère C;

et les autres influences partant de la périphérie, le système cérébro-spinal, les organes internes etc. par le caractère X, en presumant que X est stable ou ne change que très peu, alors l'on peut, pour faciliter la compréhension, établir la formule suivante:

$$T = C + L + X$$

dans laquelle C représente une grande valeur tandis que l'influence L est sujet à des variations multiples.

Pour éviter une attaque et un état incertain intermittent, il faut que C soit réduit. A présent nous avons cette réduction de C en notre pouvoir de sorte que la valeur stimulante pour l'attaque n'entre pas en vigueur.

**Medicine.** — *De l'immunité contre le phosgène.* Par ONG SIAN GWAN.  
(Communicated by Prof. E. GORTER.)

(Communicated at the meeting of January 25, 1941.)

1. On sait que le phosgène,  $\text{COCl}_2$ , un des gaz de guerre les plus redoutables, a des affinités chimiques très vives. On pourrait ainsi supposer que l'action énergique du phosgène sur les poumons donnerait naissance à une combinaison chimique nouvelle, formée par les protéines du poumon ayant été en contact avec le phosgène. Cette protéine modifiée et devenue étrangère pour l'individu intoxiqué agirait comme antigène. Cette hypothèse s'appuie sur le fait suivant: M. A. KLING et ses collaborateurs ont constaté que le phosgène, le chlore et le brome réagissent avec les stérols du poumon qui, après cette réaction deviennent hydrofuges <sup>1)</sup>).

Il y a lieu par conséquent de se demander si l'on ne pourrait arriver à vacciner les animaux contre le phosgène en les soumettant préalablement à des doses progressives de phosgène. Les résultats expérimentaux suivants, que nous avons obtenus, semblent confirmer l'hypothèse précédente.

2. *Technique expérimental.* a. L'appareil employé dans toutes ces expériences est constitué par un ballon en verre solide A, d'un volume de 35 litres (fig. 1) <sup>2)</sup>. Le col du ballon est protégé par un anneau en laiton. Un couvercle lourd en laiton B est adapté sur le col; pour assurer la fermeture du ballon on intercale un anneau en caoutchouc entre le col et le couvercle et de plus celui-ci est attaché au support du ballon par deux ressorts métalliques. Le couvercle est muni de deux robinets C et D et d'une troisième ouverture au centre où se trouve l'agitateur mécanique E. Celui-ci est formé par un disque circulaire de mica E suspendu à un fil métallique et terminé à son extrémité supérieure par une boule de fer. Pourquoi'il n'y ait aucune communication avec l'air extérieur la boule de fer est enfermée dans un tube en verre F, qui est relié au couvercle par un tube en caoutchouc. L'agitateur est actionné par un electro-aimant G ayant un mouvement de haut en bas. Ce mouvement est obtenu en reliant l'electro-aimant à un point excentrique du disque H, qui à son tour est actionné par un electro-moteur I. L'agitateur employé a une fréquence de 40 par minute. Ce dispositif est couramment employé dans notre laboratoire de physique. Enfin le robinet D est relié au moyen d'un tube détachable en caoutchouc à une pompe centrifuge J d'une grande capacité.

<sup>1)</sup> C. R. Acad. Sc. **197**, 1782 (1933).

<sup>2)</sup> Construit par notre regretté et habile constructeur M. P. J. VAN DER BAAN, qui d'une aimabilité touchante, était toujours prêt à nous rendre service.



*b. Marche d'une expérience.* Après avoir introduit l'animal dans le ballon A on attache le couvercle sur le col du ballon à l'aide de deux ressorts. On introduit le phosgène dissout dans le toluène par le robinet D,

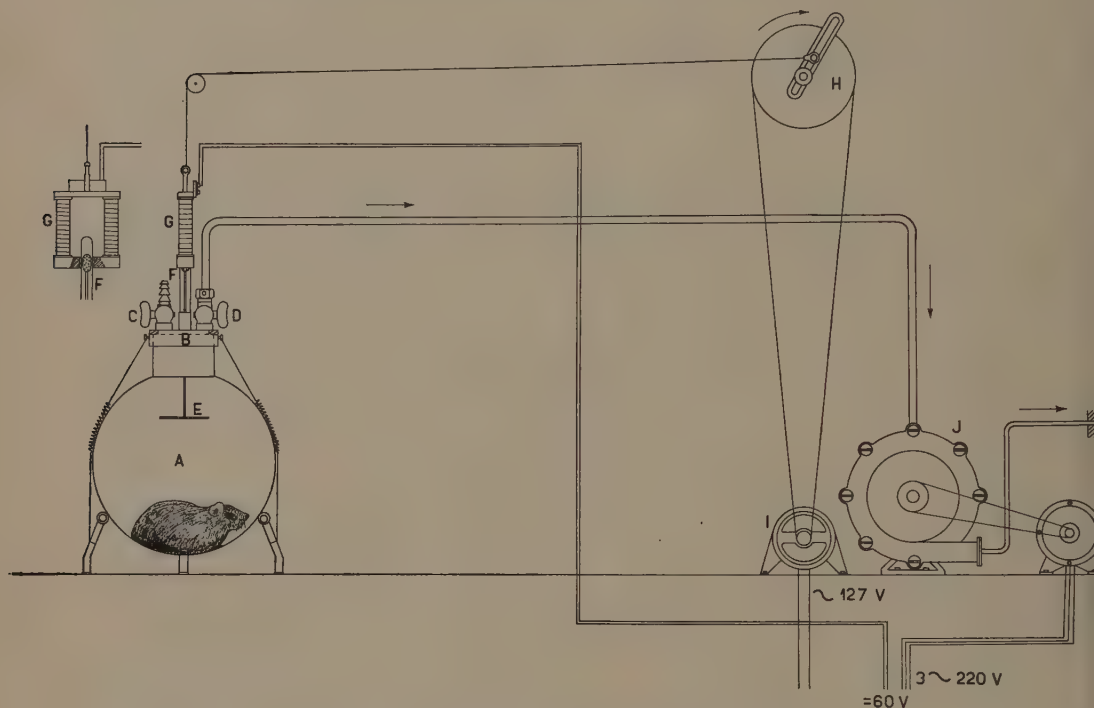


Fig. 1. Appareil pour intoxication phosgénique.

tandis que l'autre robinet C reste fermé. La solution de phosgène tombée sur le disque de mica s'évapore très vite. On ferme aussitôt après le robinet D, on déclenche le chronomètre et en même temps on fait fonctionner l'agitateur. On note la température du laboratoire, l'humidité de l'air et on fait surtout attention si pendant l'expérience l'animal reste tranquille, s'il y a émission des urines et des fèces. À la fin de l'expérience on met en communication le tube en caoutchouc avec le robinet D. Ensuite on arrête l'agitateur; au bout d'un temps déterminé on ouvre le robinet C et en même temps on fait marcher la pompe centrifuge, qui aspire le mélange air-phosgène et le jette en dehors du laboratoire. Après 4 min. on arrête la pompe centrifuge et on enferme l'animal dans une cage séparée pour l'observation. La durée de l'intoxication est exactement 15 min. pour la souris et 20 min. pour le cobaye.

*c. Solution de phosgène.* On utilise dans toutes ces expériences une solution de phosgène dans le toluène (environ 25 mg par cc)<sup>3)</sup>. On introduit dans le ballon le nombre de gouttes calculé (0,1 cc = 5 gouttes) en

<sup>3)</sup> Nous remercions MM. le Professeur J. J. BLANKSMA et J. VAN ALPHEN, qui ont bien voulu nous fournir le phosgène.

se servant toujours de la même pipette graduée au 0,02 cc. Des essais préliminaires ont montré que la quantité de toluène (1 à 10 gouttes) employée n'a aucune action nocive sur des cobayes neufs. Quoique la solution de phosgène soit gardée dans un vase de DEWAR contenant un mélange frigorifique (glace + sel marin), le titre s'abaisse en fonction du temps. Il est donc nécessaire de le déterminer souvent, surtout si le tube a été fréquemment ouvert. Le titrage est fait en décomposant le phosgène par NaOH et ensuite on détermine quantitativement les ions  $\text{Cl}^-$  libérés par la méthode de VOLHARD.

d. *Animaux employés.* Ce sont des cobayes et des souris blanches élevées par nous-mêmes. Il est important de prendre des souris d'une même souche, car les souris du commerce donnent une sensibilité variable vis-à-vis du phosgène. Les animaux sont observés pendant longtemps avant d'être employés. On constate assez souvent que les cobayes grattent leur nez et qu'ils toussent pendant l'intoxication. On observe surtout ce dernier symptôme quand les animaux ont déjà eu auparavant une dose de phosgène.

3. *Vaccination par des doses croissantes de phosgène.* On pratique la méthode classique utilisée en microbiologie: au lieu d'injecter des doses croissantes de vaccin à des intervalles déterminés on fait respirer les animaux dans un atmosphère contenant des doses croissantes de phosgène à des intervalles déterminés. Ensuite on administre au bout d'un certain temps une dose de phosgène sûrement mortelle pour les animaux non traités.

Le tableau 1 montre les doses de phosgène en mg par 70 litres d'air. Les intervalles entre deux choses consécutives sont indiqués en jours par les chiffres entre parenthèses. Pour apprécier l'état général des animaux on les pèse avant chaque intoxication. On voit que le poids augmente quand la vaccination est bien exécutée. Les doses mortelles pour les animaux témoins sont imprimées en caractères gras. On constate que les animaux après l'administration des doses de phosgène inférieures à la dose mortelle, peuvent supporter une dose mortelle de phosgène. De plus, ces animaux peuvent ultérieurement supporter une dose égale et même supérieure à la dose mortelle de phosgène. Et on peut répéter cette intoxication plusieurs fois sans que les animaux meurent.

Les animaux vaccinés et éprouvés par la dose mortelle de phosgène n'ont en général qu'une dyspnée superficielle, d'autres par contre ne montrent aucun symptôme. Très rarement on observe un œdème pulmonaire passager. Les animaux témoins mouraient tous en 24 heures, comme le tableau 3 nous montre. Parmi les témoins il y en a deux qui ne figurent pas dans le tableau 3 et qui sont morts en 24 heures d'un œdème pulmonaire après avoir reçu une dose de 5,5 mg de phosgène par 70 litres d'air.

Comme dans la vaccination microbienne on a ici aussi des échecs. Ceux-ci mouraient tous en 24 heures après une dose égale ou supérieure à la dose mortelle. A l'autopsie on constatait un œdème pulmonaire aigu exactement

TABLEAU 1.

Résultats des vaccinations contre le phosgène chez des cobayes.

No et sexe des cobayes	Poids en gr.	Doses de phosgène administrées en mg. par 70 litres d'air	Observations
7, m.	320; 310; 340; 325; 320; 340; 380	3,5(19) 1,0(11) 1,0(9) 1,5(10) 2,0(81) 1,76(11) 2,6	Cédème pulmonaire passa après la première dose de ph gène.
21, f.	354; 370; 370; 385; 420; 350; 405; 380; 519; 640; 730; 720	1,0(12) 1,0(9) 1,5(84) 1,6(8) 1,5(5) 2,0(9) 2,36(5) 2,32(45) 2,83(67) 2,72(25) 2,85(109) 2,88	Elle est en très bonne santé.
22, m.	365; 395; 430; 470; 450; 420; 390; 428; 425; 522; 580; 590; 700; 650; 660	1,0(12) 1,0(9) 1,5(84) 1,56(7) 1,5(5) 2,0(10) 2,3(18) 3,0(21) 2,88(10) 2,83 (67) 2,72(7) 3,33(48) 2,85(14) 2,85(120) 5,8	Il est mort 24h. après la nière dose de phosgène. Autop cédème pulmonaire aigu.
23, f.	315; 300; 430; 390; 390; 375; 350; 480; 485	1,0(11) 1,0(81) 1,56(8) 1,46(5) 1,98(8) 2,36(36) 2,32(80) 2,78(8) 3,32	Elle est morte 39j. plus t d'une infection secondaire: p teurellose.
25, m.	310; 340; 300; 260; 305; 295; 345; 330	1,65(9) 1,5(6) 1,98(7) 1,8(19) 3,0(22) 2,83(76) 2,78(9) 3,30	Il est mort 25j. plus tard d' maladie intercurrente.
27, f.	380; 400; 420; 353; 388; 545; 540	1,3(7) 1,8(5) 2,25(21) 1,88(11) 2,32(19) 2,78(9) 3,33	Mort 100j. plus tard d' pasteurellose.
28, m.	450; 420; 435; 457; 442; 422; 600; 610; 600; 630	1,3(8) 1,77(4) 2,25(16) 1,96(6) 2,75(15) 3,47(76) 3,64(7) 4,16(49) 2,5(14) 3,3	Il est en très bonne santé.
30, m. albinos	390; 400; 445; 457; 402; 438; 545; 460	1,28(5) 1,8(5) 1,7(14) 2,0(7) 2,8(16) 3,47(71) 3,0(11) 3,44	Il est mort 9j. après d'une pl résie et pneumonie caséuse.
32, m.	282; 275; 287; 298; 365; 375; 430; 410; 450	0,97(6) 1,8(6) 1,65(16) 1,74(63) 2,0(11) 2,56(63) 2,85(15) 2,66(105) 5,8	Il est en très bonne santé.
33, m.	298; 340; 338; 418, 400	0,97(12) 1,65(16) 1,74(63) 2,0(11) 2,56	Idem.
43, f.	535; 550; 560	1,75(5), 1,0(7) 2,85	Idem.
46, m.	300; 320; 310; 285	1,2(6) 1,32(6) 1,77(7) 2,77	Mort 5j. plus tard d'une c gestion pulmonaire.
47, f.	310; 330; 320; 330; 310	1,2(6) 1,32(6) 1,77(7) 2,29(21) 2,23	En très bonne santé.
48, m.	450; 455; 455; 470; 455	1,2(6) 1,32(6) 1,77(7) 2,29(20) 2,55	Idem.
49, f. albinos	440; 460; 480; 480; 490	1,2(6) 1,32(6) 1,77(8) 2,65(19) 2,23	Idem.

TABLEAU 1. (suite).

Résultats des vaccinations contre le phosgène chez des cobayes.

Sexe des cobayes	Poids en gr.	Doses de phosgène administrées en mg. par 70 litres d'air	Observations
m.	440; 400; 410; 420; 445	1,2(5) 1,32(6) 1,77(8) <b>2,29(19) 2,23</b>	Idem.
m.	420; 370; 360; 370; 435	1,2(5) 1,32(6) 1,77(8) <b>2,29(19) 2,55</b>	Idem.
A. m.	310; 380	2,85(14) <b>6,13</b>	Idem.
m.	340; 340; 350	1,92(2) <b>5,5(6) 6,1</b>	Idem.
f.	370; 370; 400	1,92(3) 5,03(5) <b>6,1</b>	Idem.
m.	310; 340; 350	0,96(5) 5,03(37) <b>5,8</b>	Idem.
m.	330; 350; 355	0,96(5) 5,03(37) <b>5,8</b>	Idem.
f.	400; 400	1,68(1) <b>5,67</b>	Idem.
f. nos	410; —	1,68(1) <b>5,67</b>	Idem.
m.	—; 420	4,86(3) <b>6,1</b>	Idem.
m. nos	420; 415	3,23(1) <b>5,1</b>	Idem.
m.	520; 530	4,04(1) <b>5,1</b>	Idem.
m. nos	420; 410	4,35(2) <b>5,8</b>	Idem.
f.	280; —; 324	1,24(2) 2,85(15) <b>6,1</b>	Mort 4h. après la dernière dose de phosgène: œdème pulmonaire.
f.	310; 370; 359	2,44(15) <b>6,13</b>	Mort en 24h. après la dernière dose de phosgène: œdème pul- monaire.
m.	300; 330; 315,6	3,85(6) <b>5,67</b>	Mort 2j. après: congestion pul- monaire.
m. nos	—; 390; 368,5	1,68(2) <b>6,13</b>	Mort en 24h., œdème pulmo- naire aigu.
f. nos	—; 380	1,68(3) <b>6,1</b>	Mort 5h. après: œdème pulmo- naire.

Les chiffres entre parenthèses indiquent le nombre de jours qui sépare les deux doses consécutives de phosgène. Les poids sont déterminés avant chaque intoxication phosgénique. Les doses mortelles de phosgène sont imprimées en caractères gras.



comme chez les animaux témoins. Les insuccès sont présentés en bas du tableau 1. Il est à noter que le cobaye 93 est probablement mort d'une maladie intercurrente, car à l'autopsie on ne constatait pas d'œdème pulmonaire et de plus l'intervalle entre la dose mortelle et la mort était 48 heures au lieu de moins de 24 heures.

4. Il faut remarquer que vers la fin de novembre 1940, la dose mortelle de phosgène était devenue au moins 5,1 mg par 70 litres d'air au lieu d'environ 2,2 mg en été, c'est-à-dire qu'elle est plus que doublée. On peut la constater en comparant les cobayes 80, 80A, 101, 107, 108, 110 avec les cobayes témoins 55, 45, 26, 54 et 24 dans les tableaux 1 et 3.

Avec l'aimable concours de M. K. F. WALDKÖTTER nous avons examiné la solution de phosgène dans le toluène: si le phosgène est transformé en fonction du temps. Le titrage d'après la méthode mentionnée (paragraphe 2c) donnait 24,2 mg de phosgène par cc. Ce résultat montre que la solution ne contient pas de chlore combiné, comme le prouve également le résultat négatif de la réaction de BEILSTEIN faite sur la solution après évaporation du phosgène. De plus cette solution donnait des traces d'ions  $\text{Cl}^-$  avec le nitrate d'argent. Enfin nous avons déterminé la quantité de phosgène sous forme de diphénylurée d'après le procédé de KLING et SCHMUTZ. La solution contenait 23,8 mg de phosgène par cc, valeur sensiblement égale à celle obtenu d'après la méthode employée. Les indices de réfraction déterminés à  $21,7^\circ \text{C}$ . sont  $n_D = 1,49540$  pour le toluène et  $n_D = 1,49521$  pour la solution de phosgène après évaporation du phosgène. Il n'y a donc pas de différence notable.

Il reste à savoir si la différence de température, d'humidité de l'air et de nourriture ne jouent pas un rôle. La différence de température et d'humidité n'est pas grande et la nourriture est la même, ces facteurs ne peuvent pas expliquer la différence de sensibilité des cobayes.

Il faudrait donc attribuer ce résultat à une différence de sensibilité saisonnière du cobaye vis-à-vis du phosgène.

5. *Vaccination par une dose unique de phosgène.* Non seulement les cobayes sont réfractaires à la dose mortelle de phosgène après être vaccinés par des doses croissantes de phosgène, mais ils le sont également après avoir subi une dose unique de phosgène. Les résultats obtenus chez les cobayes 80A, 97, 98, 101, 107, 108 et 110 (tableau 1) nous le montrent clairement.

6. *Apparition de l'immunité.* L'immunité s'établit 24 h. après la dose vaccinante de phosgène. En effet, aucun des quatre cobayes ( $N^0$ , 97, 98, 107 et 108, tableau 1) éprouvés 24 h. après par la dose mortelle de phosgène est mort. Le tableau 1 montre également que les cobayes qui ont reçu une dose unique de phosgène sont réfractaires à la dose mortelle de phosgène dans les jours suivants.



On pourrait également essayer la fonction suivante

$$p = \frac{aP}{100 + bP} \cdot \cdot \cdot \cdot \cdot \cdot \cdot (4)$$

Cette équation tends vers une limite, quand  $P$  croît indéfiniment

$$\lim_{P=\infty} \frac{aP}{100 + bP} = \frac{a}{b}.$$

On trouve dans le tableau 2 le poids du poumon calculé avec les formules (1), (2) et (4). Le calcul des constantes  $a$  et  $b$  est fait dans les équations (1) et (2) par la méthode des moindres carrés. On trouve ainsi pour

TABLEAU 2.  
Relation entre le poids du poumon et le poids total du cobaye neuf.

Poids des cobayes en gr. $P$	Poids du pou- mon en gr. $P_{\text{obs.}}$	Volume du poumon en cc $v$	Poids du poumon calculé avec les formules			Densité du poumon $\frac{P_{\text{obs.}}}{v}$
			$p = a + bP$	$p = aP^b$	$p = \frac{aP}{100 + bP}$	
70,9	1,55	2,5	2,11	1,57	1,81	0,62
94,05	1,60	3,4	2,21	1,81	2,11	0,47
95,6	1,30	1,6	2,22	1,83	2,12	0,81
152,0	1,30	2,6	2,46	2,31	2,60	0,50
158,5	3,20	4,0	2,49	2,36	2,64	0,80
180,0	3,80	4,8	2,59	2,52	2,77	0,79
188,4	2,90	4,6	2,62	2,58	2,81	0,63
190,0	3,40	4,25	2,63	2,59	2,82	0,80
197,0	3,90	4,2	2,66	2,64	2,85	0,93
209,5	4,10	5,9	2,72	2,72	2,91	0,69
230,0	2,90	—	2,80	2,85	2,99	—
244,5	2,80	6,0	2,87	2,94	3,04	0,47
291,9	2,60	3,2	3,08	3,21	3,18	0,81
294,0	3,05	5,3	3,08	3,23	3,19	0,58
316,2	2,05	2,6	3,18	3,35	3,25	0,79
492,0	3,90	4,7	3,95	4,18	3,53	0,83
Total	44,35	59,65				10,52
Moyennes	2,77	3,98				0,70

l'équation (1):  $a = 1,7997$  et  $b = 0,00437$ . En prenant comme valeurs  $a = 1,8$  et  $b = 0,0044$  on obtient des résultats équivalents. Les constantes de l'équation (2) sont:  $a = 0,1825$  et  $b = 0,5053$ . Dans l'équation (4) les constantes  $a$  et  $b$  sont déterminées par la méthode des groupements. On trouve  $a = 4,4837$  et  $b = 1,0652$  et la limite cherchée.

$$\lim_{P \rightarrow \infty} \frac{aP}{100 + bP} = \frac{4,4837}{1,0652} = 4,2.$$

11. *Signification des formules.* Pour savoir si les valeurs calculées avec les formules (1), (2) et (4) correspondent aux valeurs trouvées par l'observation on calcule le coefficient de corrélation  $\varrho$ .

$$\varrho^2 = 1 - \frac{\sum (p_{\text{obs.}} - p_{\text{calc.}})^2}{\sum (p_{\text{obs.}} - \bar{p}_{\text{obs.}})^2}.$$

La signification de la valeur trouvée  $\varrho$  pour des observations peu nombreuses peut être évaluée par la méthode de R. A. FISCHER <sup>5)</sup>.

$$t = \frac{\varrho}{\sqrt{1 - \varrho^2}} \sqrt{n - 2}$$

Si la probabilité pour que,  $t$  soit supérieure à la valeur trouvée est inférieure à 5p. 100 on peut dire que la formule empirique est significative. On trouve ainsi pour  $n - 2 = 14$ , les valeurs suivantes

formule (1)	$\varrho = 0,49$	$t = 2,104$	probabilité cherchée $\cong 0,06$
„ (2)	$\varrho = 0,52$	$t = 2,271$	„ „ $\cong 0,04$
„ (4)	$\varrho = 0,59$	$t = 2,745$	„ „ $\cong 0,02$

La formule (1) est donc peu ou pas significative, la meilleure approximation est obtenue par la formule (4).

12. *Expression du poids de poumon œdémateux en fonction du poids total de cobaye mort par intoxication phosgénique.* La figure 2 et le tableau 3 montrent la relation entre le poids du poumon œdémateux et le poids total du cobaye mort par intoxication phosgénique. On voit en même temps les courbes calculées d'après les équations (1), (2) et (4). Dans le graphique sont également placées deux observations désignées par des carrés, qui sont venues après la détermination des courbes. On constate que les formules empiriques (1), (2) et (4) donnent aussi une bonne approximation pour le poumon œdémateux. En effet on trouve pour  $n - 2 = 17$ ,

formule (1)	$\varrho = 0,72$	$t = 4,102$	probabilité cherchée $< 0,001$
„ (2)	$\varrho = 0,74$	$t = 4,549$	„ „ $< 0,001$
„ (4)	$\varrho = 0,73$	$t = 4,430$	„ „ $< 0,001$

<sup>5)</sup> R. A. FISHER, Statistical methods for research workers, London and Edinburgh, Oliver and Boyd, 198 (1938).



TABLEAU 3.

Relation entre le poids du poumon oedémateux et le poids total du cobaye mort par intoxication phosgénique. Rapport du poids de poumon oedémateux à celui de poumon normal; densité du poumon oedémateux.

No. et sexe des cobayes	Doses de phos-gène administrées en mg. par 70 litres d'air	T	Poids des cobayes en gr. P	Poids du poumon en gr. $p_{obs.}$	Volume du poumon en cc. $v$	Poids du poumon normal calculé $p_{calc.}$	Rapport $\frac{p_{obs.}}{p_{calc.}}$	Poids du poumon oedémateux calculé avec les formules			Densité $\frac{p_{obs.}}{v}$
								$p = a + bP$	$p = aPb$	$p = \frac{aP}{100 + bP}$	
* 20, f.	~ 2,4	< 24 h.	240,0	5,2	6,25	3,03	1,72	6,71	6,62	6,24	0,83
* 12, f.	~ 4,2	< 24 h.	279,5	8,0	—	3,15	2,54	7,41	7,35	7,17	—
* 15, f.	~ 2,8	3,5 h.	280,0	8,8	9,25	3,15	2,79	7,42	7,35	7,18	0,95
* 55, albinos	2,3	< 24 h.	283,6	6,6	7,3	3,16	2,09	7,48	7,42	7,26	0,90
* 45, f.	2,85	4 h.	285,7	7,1	9,0	3,17	2,24	7,52	7,46	7,31	0,79
* 26, f.	2,15	8 h.	289,5	8,4	10,5	3,18	2,64	7,59	7,52	7,40	0,80
* 13, f., albinos	~ 4,2	2 h.	290,0	6,5	—	3,18	2,04	7,60	7,53	7,41	—
* 8, f., albinos	~ 6,0	< 24 h.	290,0	6,6	—	3,18	2,08	7,60	7,53	7,41	—
* 14	~ 3,5	6,5 h.	300,0	7,7	—	3,21	2,40	7,77	7,71	7,64	—
* 54, m; albinos	2,3	< 24 h.	302,5	7,8	9,8	3,21	2,43	7,82	7,75	7,69	0,80
* 9, m.	~ 6,0	2 h.	310,0	9,6	—	3,23	2,97	7,95	7,88	7,86	—
79, f.	1,24; 2,85; 6,1	4 h.	324,0	9,5	11,0	3,26	2,91	8,20	8,12	8,18	0,86
* 10, m.	~ 4,0	< 24 h.	325,0	7,9	—	3,27	2,42	8,21	8,14	8,20	—
80, f.	2,44; 6,13	< 24 h.	359,0	10,0	12,3	3,34	2,99	8,81	8,71	8,95	0,81
* 109, f., albinos	5,1	4 h.	(359,7)	(9,55)	11,7	3,34	2,96	(8,83)	(8,76)	(8,96)	0,82
99, m., albinos	1,68; 6,13	< 24 h.	368,5	6,9	9,5	3,35	2,06	8,98	8,86	9,16	0,73
100, f., albinos	1,68; 6,1	5 h.	380,0	8,0	9,2	3,38	2,37	9,18	9,05	9,40	0,87
* 102, f.	5,67	< 24 h.	439,5	11,9	15,8	3,47	3,43	10,23	9,99	10,65	0,75
29, m.	1,3; 1,8; 2,25; 3,0	< 24 h.	481,5	13,05	18,3	3,52	3,71	10,97	10,63	11,51	0,71
* 24, m.	2,82	3,75 h.	572,25	10,5	12,0	3,62	2,90	12,57	11,95	13,27	0,87
22, m.	voir tableau 1	< 24 h.	(636,05)	(13,3)	18,2	3,67	3,62	(13,70)	(12,84)	(14,45)	0,73
Total			160,05								12,22
Moyenne			8,42								0,81

\* Cobayes témoins.

T = Temps écoulé entre la dose mortelle (première ou dernière dose) de phosgène et la mort du cobaye.

Les valeurs entre parenthèses sont obtenues après la détermination des courbes.

Dans les cas considérés les trois formules sont donc significatives. Les valeurs dans la colonne 7 du tableau 3 sont calculées à l'aide de la formule (4) avec les constantes  $a = 4,4837$  et  $b = 1,0652$  obtenues pour le poumon normal. La colonne 8 donne le rapport du poids de poumon œdémateux à celui de poumon normal calculé. L'œdème pulmonaire est d'autant plus prononcé quand ce rapport est plus élevé.

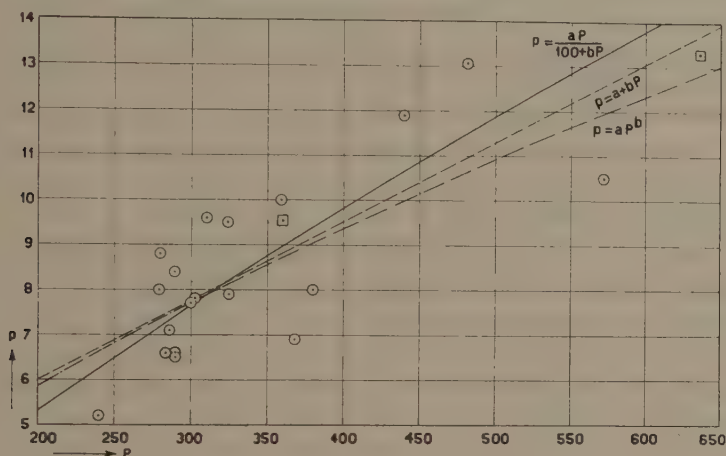


Fig. 2. Relation entre le poids du poumon œdémateux  $p$  et le poids total du cobaye  $P$  mort par intoxication phosgénique. Les carrés représentent des observations obtenues après la détermination des courbes.

Les constantes  $a$  et  $b$  pour le poumon œdémateux sont les suivantes: formule (1),  $a = 2,4799$ ,  $b = 0,01764$ ; formule (2),  $a = 0,1605$ ,  $b = 0,6788$ ; formule (4),  $a = 2,853$ ,  $b = 0,040238$  avec une limite

$$\lim_{P=\infty} \frac{aP}{100 + bP} = \frac{2,853}{0,040238} = 70,9$$

valeur très élevée, qui sans doute ne sera pas atteinte si l'on fait les expériences avec le cobaye. Ceci prouve que la formule (4) est bonne.

13. *Densité du poumon œdémateux.* Pour savoir si la différence entre la densité moyenne du poumon œdémateux ( $\bar{d} = 0,81$ ) et celle du poumon normal ( $\bar{d}' = 0,70$ ) est significative, on détermine la valeur de  $t$ , qui exprime le rapport de la différence  $\bar{d} - \bar{d}'$  à l'écart type de la différence. Si la probabilité des valeurs supérieures à la valeur observée  $t$  est inférieure à 5 p. 100, on dit que la différence est réelle.

On trouve ainsi pour  $n = 28$ ,  $t = 2,634$  et la probabilité cherchée serait égale à 1 p. 100. On en conclut que la différence de densité moyenne du poumon normal et celle du poumon œdémateux est réelle.

14. *Comparaison des résultats obtenus avec les statistiques de la guerre de 1914—1918.* A l'aide des résultats obtenus par nos expériences on peut

faire des déductions importantes. Il est à prévoir que la mortalité des combattants, qui ont subi une première attaque de phosgène ou d'autres gaz de guerre, serait beaucoup plus élevée que celle obtenue après une deuxième, troisième, etc. attaque du même gaz de guerre.

D'après O. MUNTSCH <sup>6)</sup> la mortalité causée par les gaz de guerre diminue vers la fin de la guerre. L'auteur l'explique par l'emploi et l'amélioration du masque. Il est raisonnable d'y ajouter un deuxième facteur: l'immunité que les hommes gazés ont obtenu après un ou plusieurs contacts avec les gaz toxiques. Malheureusement on ne peut pas savoir d'après les statistiques publiées après la guerre combien parmi les hommes gazés ont déjà été en contact avec les gaz toxiques. De plus on sait qu'on employait souvent un mélange de plusieurs gaz toxiques, ce qui complique les résultats et par conséquent difficile de conclure.

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<sup>6)</sup> O. MUNTSCH, Leitfaden der Pathologie und Therapie der Kampfgaserkrankungen, Georg Thieme, Leipzig, 14 (1934).

**Zoology.** — *Observations on a special manner of feeding of a species of Diffflugia (Diffflugia rubescens PENARD). By H. R. HOOGENRAAD and A. A. DE GROOT. (Communicated by Prof. J. BOEKE.)*

(Communicated at the meeting of January 25, 1941.)

(*Rhizopoda and Heliozoa of the Netherlands. VIII.*)

The species of *Diffflugia* belonging to the thecamoebae *Rhizopod* genus feed in general by ingesting "ready" food: all kinds of smaller organisms and particles of sapropelium, which are digested in food-vacuoles. As is known this is the usual manner of feeding of the vast majority of marine and freshwater *Rhizopoda*; by the side of it there are as variants cases of intracellular symbiosis (e.g. *Amoeba viridis*, *Paulinella chromatophora*, *Hyalosphenia papilio*, species of *Amphitrema*), in which the ingestion of ready food recedes into the back-ground or is entirely absent, and a number of representatives of the *Vampyrella* group (species of *Vampyrella*, *Vampyrellidium*, *Hyalodiscus*, *Leptophrys*), which feed on the living cell contents of various species of algae; they ingest these contents, after first puncturing the wall of the alga cell they have chosen for their prey.

This peculiar manner of feeding was discovered in 1865 by CIENKOWSKY in *Vampyrella lateritia* (= *Spirogyrae*), which lives on the cell contents of various *Spirogyra* species and other filamentous algae belonging to the group of the *Conjugatae*. Afterwards it has also been found in related species, which all empty in the same manner the cells of *Conjugatae*, occasionally (*Vampyrellidium*) also those of *Cyanophyceae*. In 1927 one of us (HOOGENRAAD) described a form (*Leptophrys*), which makes the cells of a species of *Desmidiaceae* (*Closterium Dianae*) its prey; a second case of feeding on the cell contents of a species of *Closterium* (*Closterium intermedium*) is mentioned by POISSON and MANGENOT (1933) in a *Rhizopod*, which they called *Vampyrella Closterii*. Besides, MARGARETE ZUELZER (1927) discovered an *Amoeba* (*Amoeba biddulphiae* n. sp.), which in an entirely similar way feeds on the cell contents of a marine *Diatomea*, *Biddulphia sinensis*. In all these cases the *Rhizopods* showing the aberrant manner of feeding belonged to the group of the *Gymnamoebae*, hence of the naked forms, that are not provided with a theca.

Since then it appeared, however, that this manner of feeding is not confined to this group. In 1935 STUMP discovered it in North America in a few species of thecamoebous *Rhizopoda*, belonging to the interrelated genera *Diffflugia*, *Pontigulasia* and *Lesquereusia*: just like the species of the *Vampyrella* group these animals feed on the cell contents of the species



of *Conjugatae*. At the same time (1935) we mentioned some preliminary observations of a species of *Diffflugia* (*D. pulex* PENARD), which seems to ingest the cell contents of filamentous *Cyanophyceae*; at the time we were unable, however, to state further particulars. Now, during a short stay at the Biological Station at Wijster, in July and August 1940, we collected there a number of samples of sphagnum and sapropelium; one of the latter appeared to contain a rich population of another species of *Diffflugia*, viz. *D. rubescens* PENARD, which showed the same manner of feeding, with an object for its prey, however, different from the *Diffflugia* species of STUMP. This case had been unknown so far; below we submit a short report of our observations.

Among the species of the genus *Diffflugia* *D. rubescens* (Fig. 1) occupies a somewhat separate place owing to two characteristics, by which it varies from the other species of the genus. As an independent species it was described for the first time by PENARD in 1891 from material from the Rocky Mountains, which material contained exclusively encysted, and no living specimens. Previously (1879) it had probably been observed already by LEIDY, likewise in North America, but had been considered as an aberrant variant of *D. oblonga* EHRBG. (= *D. pyriformis* PERTY). Since then it has also been recognised in various places in Europe; in the Netherlands we know it from a dozen of localities, all of them in sphagnum, or at least with sphagnum contact.

Of the two characteristics differentiating *D. rubescens* from the other species of the genus one concerns the shell, the other the cytoplasm. The shell is not colourless, but a lighter or darker brownish yellow and rather transparent. While the pseudo-chitinous material seems to be more strongly developed than in the other species of *Diffflugia*, owing to which the thickness of the wall of the shell is greater than usual, the covering with xenosomata is mostly rather scarce, which, also thanks to the transparency of the shell, causes the cytoplasm to be usually well perceptible through the wall of the shell. Occasionally, however, the shell bears, mostly be it in small numbers only, enormous *Diatomeae* cells and/or grains of silica. The mouth is round with a more or less crenate inner-edge.

The cytoplasm itself, just as in other species of *Diffflugia*, is colourless; it contains, however, a smaller or larger number of glossy, spherical elements, coloured a bright orange-red, which are sometimes heaped up locally, on other occasions are scattered throughout the cytoplasm, and which also penetrate into the basis of the pseudopodia, occasionally even into the top of them. In spite of the fact that PENARD calls them "grains" the always rather pure spherical shape seems in our opinion to point rather to the liquid state of aggregation; we would therefore prefer to consider them as drops. Never did we see two or more of them merge, however; if, therefore, they are in a liquid condition indeed, it is possible that just as in the case of the disperse elements of an emulsion a surface layer of a somewhat different consistence will prevent them from joining.

The size of the shell seems to be very variable. PENARD (1891) states a length of 30—35  $\mu$ , but reports afterwards from the environs of Geneva two forms with a length of 58—60  $\mu$  and 83—90  $\mu$  respectively. So far we were able to ascertain for populations of very different origin also a length of 65—95  $\mu$ . Now the Wijster population seems to be the largest ever observed; the length of the shell varied from 60—140  $\mu$ , the breadth from 40—70  $\mu$ .

The scanty information about this species in the half century elapsed since its discovery refers to the morphology; little if anything was known about its manner of living. Likewise, data about the nature and the ingestion of food are wanting. Now at an earlier date already, in sphagnum of Vriezenveen, we had come across some specimens of this species with dirty-green inclusions in their cytoplasm, pointing to chlorophyll feeding; to this we did not pay special attention, however, at the time. In the Wijster sapropelium sample, containing the *Diffflugia rubescens* population, there were also some species of *Desmidiaceae*, *inter alia* some species of *Closterium*, viz., according to the determination of Dr. BEIJERINCK, Director of the Wijster Biological Station, *Closterium Ulna*, *intermedium* and *striolatum*, the former general, the latter two rarer. In the early part of October 1940 our attention was drawn by the fact that living specimens of *Diffflugia* were not infrequently seen lying with their mouth against a sound or empty *Closterium* cell, while, when the *Closterium* cell shifted, e.g. by letting a drop of water flow under the cover glass, the *Diffflugia* individual also moved, the mutual position, however, showing no change whatever.

A short time after a *Diffflugia* individual was found in the same position with respect to a *Closterium* cell, which was not sound, but for the greater part empty; only in the cell tops there were two small, elliptical masses of chlorophyll, evidently remnants of the two chromatophores (Fig. 2 a). The animal was just beside the middle of the alga cell. A further examination showed that on the left and on the right in the cell there lay stretched out a *Diffflugia* lobopodium, enveloping with its top the remnants of the chromatophores. At the same time it appeared that the lobopodia were not at rest, but were slowly drawn in with the remnants of the chromatophores. While the latter gradually lost their outlines in the lobopodium and faded away as it were (Fig. 2 b), they ultimately disappeared in the *Diffflugia* shell; a moment after also the two lobopodia were drawn in. After some time the animal abandoned the cell. In the place where it had been attached the cell-wall showed an irregularly formed hole with fibrously frayed edges. It was not difficult to find in the same preparation another number of empty *Closterium* cells with similarly formed holes. (Fig. 3, 12—14).

The next day the ingestion of the contents of a *Closterium* cell by a *Diffflugia* could be observed from beginning to end, an observation which was repeated numerous times in the course of the following weeks (Fig. 4). On the whole the process passed off uniformly; below we are giving an

account of the case first observed as reported just now, after which we intend to give some variants and supplementary information.

When the animal was discovered (2.30 p.m.) it lay against a *Closterium* cell in the manner as described above, this time, however, not in the proximity of the middle, but close to one of the extremities, at about  $\frac{1}{7}$  of the total length of the cell ( $400\ \mu$ ) distant from it. The contents of the *Closterium* cell were, as far as we could ascertain, still completely unimpaired. At 2.40 p.m. we let a large drop of water flow under the cover glass, through which the *Diffflugia-Closterium* system shifted, it is true, but the mutual position did not change.

2.45 p.m. The chromatophore nearest to the *Diffflugia* is gradually beginning to recede from the wall — plasmolysis — under clear signs of disintegration and subsequent aggregation; the terminal vacuoles with their crystals are still intact, the latter still showing the normal Brownian movement. No *Diffflugia* pseudopodia are perceptible in the *Closterium* cell.

3.— p.m. Under distinct signs of progressive disintegration also the second chromatophore streams into the neck of the *Diffflugia*: in the neck two green, parallel streams of chlorophyll are now visible, which gradually disappear in the plasma of the *Diffflugia*; the chromatophores have lost their normal, round form and are twisted wave-like; in the *Closterium* plasma large vacuoles appear.

3.15 p.m. Also the two terminal vacuoles with the crystals are beginning to move; the twisted edges of the chromatophores stretch themselves, regaining a rounder form. All the time little change is to be seen in the shell cavity of the *Diffflugia*; only, it strikes us that the cytoplasm, which at first filled the space almost entirely, has now receded some distance from the fundus wall. There are no epipods to be seen. In the apparently very dense *Diffflugia* plasma the green colour of the *Closterium* chromatophores is becoming visible among the orange-red pigment drops of the *Diffflugia*.

3.36 p.m. From one of the chromatophores a piece snaps near the end, its position, just as that of the crystal lying behind it, remaining unchanged. The movement of the two chromatophores goes on normally, at the rate of abt.  $2\ \mu$  a minute. In the distal, empty part of the cell halves of the *Closterium* no pseudopodia are visible.

4.03 p.m. One chromatophore, except the isolated piece, has now disappeared entirely in the neck of the *Diffflugia*; the second is still abt.  $120\ \mu$  long, the distal end being still abt.  $140\ \mu$  away from the terminal vacuole, which has hardly moved yet.

4.30 p.m. In the two parts of the *Closterium* cell *Diffflugia* pseudopodia are becoming visible, extending distally, one of them ingesting the isolated remnant of the chromatophore.

4.39 p.m. This remnant disappears into the neck of the *Diffflugia*. A part of the second chromatophore is carried along by the pseudopodium, extending distally; the latter reaches the crystal — the terminal vacuole itself is no longer discernable — envelops it and recedes with the remnant of the



chromatophore and the crystal; both parts of the cell contents disappear into the neck of the *Diffflugia*.

5.— p.m. The *Diffflugia* pseudopodia, which are still stretched and which fill the two cell halves almost entirely, are gradually beginning to withdraw; the entire contents of the *Closterium* cell have now been ingested in the *Diffflugia* plasm.

5.25 p.m. The pseudopodia have been entirely drawn in, the *Closterium* cell being completely empty; the animal does not yet abandon the cell, however.

5.48 p.m. Again two pseudopodia are becoming visible in the empty cell, searching the wall gropingly, as it were.

6.10 p.m. Also these pseudopodia are drawn in, through which the cell is empty again.

6.25 p.m. The *Diffflugia* individual abandons the *Closterium* cell; in the wall of the latter the hole made by the animal is clearly visible.

As was already observed the course of the process of food ingestion of *Diffflugia rubescens* is in the main rather uniform. Of the variants the following may be mentioned.

The point of attachment of the *Diffflugia* individual can be very different. Sometimes it is close to the middle of the cell, on other occasions close to one of the extremities. Also attachment exactly in the middle — the point of the least resistance — was observed occasionally, but seems to be rare. On some occasions we found an empty cell with two holes, afterwards also a few times a sound cell, which was attacked by two individuals of *Diffflugia* simultaneously. In one of these cases the two animals were at almost exactly opposite points of the alga cell.

In most cases the entire contents of the *Closterium* cell will be ingested and in such a manner that the two chromatophores with the cytoplasm and the nucleus gradually disappear in the shell of the animal. In this process a disorganization of the chromatophores takes place, it is true, but they remain whole. In other cases the chromatophores break into a number of pieces, which are separately and one after another enveloped and ingested. Occasionally it was observed that one or more of these fragments remained behind in the *Closterium* cell; sometimes this was likewise the case with one of the gypsum crystals.

A peculiar variant in the treatment of the *Closterium* cell is shown in Fig. 5. When the animal with the alga was discovered the latter was still almost entirely sound. The *Diffflugia* individual had attached close to the middle of the cell; at this point the cell had a crack, however, the two parts being at an angle of abt.  $120^\circ$ ; the cracked part of the cell had partly penetrated into the neck of the *Diffflugia*. The process of ingestion took place in the normal manner; in the course of it the angle formed by the two halves of the cell now became smaller (to abt.  $80^\circ$ ), now larger (to abt.  $120^\circ$ ). After 50 minutes ingestion was completed and the animal released the cell; the empty cell remained cracked and showed the hole in the wall made by the *Diffflugia* precisely at the crack (Fig. 5 b). The *Closterium* cell in



question had a total length of abt.  $320\ \mu$  by a breadth of only  $10\ \mu$ ; maybe, therefore, the thickness of the wall was also slight, through which the cell was apt to crack at the point where the animal attacked it.

On another occasion again, during the process of ingestion, which for the rest was on the usual lines, one of the chromatophores broke into a number of pieces, which separated. One of them remained in the distal end of the cell half, but was gradually dissolved by a pseudopodium into small elements, the green colour of which could hardly be distinguished or not distinguished at all, after which these elements were carried by the pseudopodium to the *Diffflugia* individual.

In various cases the emptying of a *Closterium* cell, either entirely or partly, was observed, in which action no pseudopodium penetrated.

In the immediate proximity of a *Diffflugia* individual that was discovered after it had emptied a *Closterium* cell and had already abandoned it, there lay a number (abt. 20) of dark-brown grains of  $10\text{--}15\ \mu$  diameter, which gave one the impression as if they had been excreted by the animal. Afterwards these particles were also more than once seen in the cytoplasm of the animal itself, and finally it was observed — so far but once — that an individual disposed of a clump of these grains by excretion. We intend to return to the significance of this later on.

Occasionally an animal was taken in the act of attacking a *Closterium* cell (Fig. 6 a, 7, 11). Then a plug of animal plasm was clearly visible between the mouth and the wall of the plant cell. Afterwards, when contact became closer this plug disappeared again, but a mass of plasm became visible on the other side of the cell, which was evidently held from below.

The time required for the process of ingestion seems to vary a good deal. One case was over in 40 minutes; in another case a *Diffflugia* was found in contact with an entirely sound *Closterium* cell at 6 o'clock p.m., only about half of the latter's contents being ingested the next morning at 10.30, while the ingestion was not completed until noon.

As was observed before, the place of the hole made in the wall of the *Closterium* cell by the *Diffflugia* individual, varies. During ingestion it is not visible, but all the more visible in empty cells. The form, too, varies; it is usually irregularly round, elliptic or quadrangular; the size (i.e. the greatest measurement) is mostly  $10\text{--}20\ \mu$ . (Fig. 3, 12—14). The edge is never completely smooth, but always more or less irregularly crenate or corrugate, or drawn out in fibrous frays. Occasionally (Fig. 3 c) there hung beside the hole a shred of the cell wall, probably the fragment of the wall that had formerly been in the place where there was now a hole (vide p. 223).

A few times empty cells with two holes were found, and afterwards also a sound cell, to which two *Diffflugia* individuals had attached themselves at the same time; this cell was found back the next day with two holes in the wall. Finally, in a similar case, the attack on a cell by two individuals simultaneously could also be observed from beginning to end.

These individuals had attached themselves to the cell wall almost exactly opposite each other. The result was that one of them secured the whole contents of the cell but for some remnants, which remained in the cell, whereas the other did not get anything.

About the form of the holes made by *Vampyrella* and related species in the cell wall of the algae attacked, only incomplete and contradictory data are available.

CIENKOWSKY speaks of a large, not sharply outlined, hole. KLEIN is altogether silent about it, but in one of his figures pictures an alga with a small, broad, elliptical hole with smooth edges. According to SCHERFFEL the animal makes "a beautiful round hole"; LLOYD calls the hole "clean cut"; MIHAÉLOFF describes it as a star-shaped form. The holes made by *Diffflugia* c.s. in the wall of the *Spirogyra* cells always have, according to STUMP, the form of tears or rents, and never the round form of the holes made by *Vampyrella*. Finally, POISSON and MANGENOT are quite positive when they say that the wall of the *Closterium* cell is cut open by their *Vampyrella Closterii* according to a semi-circular line; from the picture it appears that in this process a fragment of the cell wall is loosened and turned back like a sort of lid. (Fig. 3g).

The nature of the process by which *Diffflugia* punctures the wall of the *Closterium* cell is still uncertain. Originally (CIENKOWSKY and others) the opinion was held that in the *Vampyrella* group this process was of a typically chemical nature, i.e. that by the excretion of matter of a perhaps enzymatous nature the animal dissolves the cell wall locally. Later observers (PENARD, ENTZ, ZUELZER, LLOYD, GOBI, POISSON et MANGENOT, STUMP) have made it probable, on the ground of observations of various objects, that partly, at least, also mechanical factors may play a part in it. This is very evident indeed in *Leptophrys elegans* (HOOGENRAAD 1927), which literally cracks and breaks in two the cells of *Closterium Dianae*, ingests the contents, subsequently to eject the two halves of the cell wall. Also the observations in the case of *Diffflugia rubescens* seem to give support to this view. Above it was already pointed out that the holes made by this animal have edges, the appearance of which suggests indeed a tearing open of the cell wall. Occasionally it was observed, as is also shown by POISSON and MANGENOT in a picture of the *Closterium* attacked by their *Vampyrella Closterii*, how as it were a piece of the cell wall is detached, though not quite separated from it, so that it hangs down from it as a rag. Particularly the case described on p. 221 of the specimen of the *Closterium* cell that was cracked in the ingestion, points to the probability that mechanical factors also play a part; it may be imagined that during the sucking action the cell cracks at the point of the hole owing to suction.

If one asks oneself how such a deviating manner of feeding as the ingestion of the cell contents, after the cell wall has been punctured, may have arisen, the following seems possible. At any rate our starting point must be the usual manner of feeding by envelopment and ingestion in

vacuoles of the particles of food. Also in some forms of the *Vampyrella* group (*Vampyrella* spec. div., *Leptophrys*) this occurs by way of exception, whereas this is the rule in the species of the genera *Amoeba*, *Diffflugia*, *Pontigulasia* and *Lesquereusia*. Also *Leptophrys elegans* ingests the *Closterium* cells in its cytoplasm, before they are broken in two; at the same time the living contents are then drawn out. To this may correspond as an extreme case that the isolated cells (*Closterium*) or the cells living in cell groups (*Spirogyra* etc.) are no longer ingested, but opened and emptied. In this connection an observation made by STUMP is of importance. For he saw that, when filaments of *Mougeotia* were attacked by *Lesquereusia spiralis* at the end, entire cell groups would be taken in through the mouth in the animal plasma and there both wall and contents were consumed. This observation affords at the same time an argument for the presence of cellulases, which, if present, might act locally in the normal manner also in the puncturing of the cells and thus might prepare the play of mechanical forces.

A striking characteristic of many *Sarcodina* which feed in the way we have described is the colour of their plasma, in which red tints, scattered diffusely or bound to granules, dominate, and are only mixed with green colours after an abundant ingestion of chlorophyllous food. When other elements take the place of chlorophyll, such as *Diatomeae*, *Cyanophyceae*, *Ciliata*, grains of starch, the red colour is lacking or at any rate less strongly developed. It is generally assumed that this pigment is caused by carotinoid components of the chlorophyll. According to POISSON and MANGENOT the pigment in the *Vampyrella Closterii* described by them is bound to globular elements — drops — of abt.  $1.5\ \mu$  diameter, which reduce osmic acid and dissolve in strong sulphuric acid, the orange-red colour of the pigment passing into a bluish green during the process. This red colouring of the plasma is nearly always lacking in *Diffflugia rubescens*. In nearly all cases we have been able to ascertain, quite in agreement with PENARD, that the plasma itself of the animal is colourless, or rather shows the peculiar greyish blue tint, which it has in most *Rhizopoda*. Only, in a very single case, we think we have seen, diffusely scattered in the plasma, very small reddish granules, imparting a faint red tint to the plasma body in question. As we have already observed above the plasma of *Diffflugia rubescens* surrounds a varying number of bright orange-red coloured drops, which are either more evenly scattered, or heaped up more locally and often penetrate into the pseudopodia to a certain distance, occasionally even to the top of them. As regards their habitus they strongly remind one of the globular, usually pale yellow, sometimes also orange coloured elements, occurring in the plasma of a problematic Rhizopod (*Diplophrys*) and an equally problematic *Heliozoon* (*Elaeorhanis*), which, though on doubtful grounds, are taken for bodies of a fatty nature. At any rate these plasma elements in *Diffflugia rubescens* do not seem to be directly connected with the chlorophyll nature of the food ingested.



We have never observed "ready food" in the plasm of these *Diffflugia*, nor is it mentioned by other observers. Only the green inclusions originating from the chlorophyll we had seen before already, without being able then to account for their nature. The fact that the drops of pigment, which are so characteristic of *Diffflugia rubescens*, are lacking in the species of the genera *Diffflugia*, *Pontigulasia* and *Lesquereusia*, which feed in an entirely similar way on chlorophyllous food, also tells in favour of the independence of these drops of possible chlorophyllous components.

The observations made of *Diffflugia rubescens* corroborate the existing view that the *Rhizopoda* which feed on the cell contents of various kinds of algae are on the whole strong specialists. In the sapropelium sample which contained the population various groups of algae were represented more or less generally, among which *Diatomeae*, *Protococcales*, *Conjugatae* and *Desmidiaceae*. *Diffflugia rubescens* confined itself in its ingestion to the three species of the genus *Closterium* just mentioned, without, as far as we could ascertain, showing any preference, however, for any of these three species. In the same material we very regularly came across individuals of *Diffflugia acuminata*, which feed only on the cell filaments of a filamentous alga of very frequent occurrence. If this specialization corresponds to a similar one of the enzymes that are perhaps produced, and the latter again to a difference in the chemical nature of the wall of the alga cells, is unknown to us.

This does not imply that *Diffflugia rubescens* might not show adaptation to other food in a different environment, as this also occurs with other Protozoa — cf. e.g. *Dinamoeba mirabilis* (DE GROOT, 1936); this was, however, not the case in our material.

Many representatives of the *Vampyrella* group encyst after ingestion, in order to digest the food taken in the nutritive cyst that has been formed. Often a division into 2—4 daughter animals takes place in the cyst, which leave the cyst as active individuals, while a number of brownish red remnants — partly digested chlorophyll? — are left behind. Something like it was not observed in *Diffflugia*; there are, however, also cysts known of this species and in some samples even fairly numerous; so far it has not appeared, however, that they are in any way connected with the ingestion, while the division, as far as it is known, is an ordinary binary division in an active state. As it was already stated above, however, brownish-red grains appear in the protoplasm of *Diffflugia* after ingestion, which grains are ejected after some time. Obviously, in these bodies analogues are seen of the remnants left behind in the cyst of *Vampyrella* and others, and the bodies are considered as waste products of the chlorophyll that cannot be digested any further.

There also seems to exist some uncertainty about the part played by the pseudopodia in ingestion. Earlier observers (ZOPF, DANGEARD, ROSEN) are of opinion that in *Spirogyra* the cell contents are as it were fished out from the cell by ramified pseudopodia. Other observers deny this and



in this form it is indeed not correct. But also the view of LLOYD, viz. that *Vampyrella* does not send a single protoplasm element into the cell is not likely in this case and in other cases surely not true. The most striking case of pseudopodial activity is certainly the one described by HOOGENRAAD (1907 b) of *Hyalodiscus rubicundus*, which, after opening and emptying a cell in the normal manner by means of a pseudopodium put out through the space of this cell, which pseudopodium has a form which normally never occurs in this species, often punctures the transverse walls of the neighbouring cells and thus also ingests their contents. Likewise, it is said by STUMP that *Lesquereusia* sometimes puts out pseudopods into the cells of the *Conjugatae* and thus ingests isolated pieces of the chromatophores which had remained fixed to the cell wall. The same phenomenon was observed by us in *Diffflugia rubescens* (vide p. 220). However, as we remarked before, we have also been able to observe various cases of ingestion in which no pseudopodium of *Diffflugia* penetrated into the cell and remnants of chromatophores were left behind in the *Closterium* cell. With *Diffflugia rubescens* the activity of pseudopods in ingestion therefore does not seem to be essential, which may be connected with the peculiar processes that take place in the interior of the *Closterium* cell.

Indeed, in many cases we were able to observe in the *Closterium* cell, attacked by *Diffflugia rubescens*, phenomena, which, as far as we can ascertain, are not reported by any of the earlier observers, and which may throw somewhat more light on the mechanism of ingestion of the cell contents (Fig. 8). Whereas in a sound *Closterium* cell the chromatophores lie very close to the cell wall, they will soon begin to recede from the wall in a cell that is being attacked, in which process the rather straight outline of the chromatophores passes into a corrugated line. Soon vacuoles appear to develop in the sinuous curves, which, growing in size constrict the chromatophores at various points; the chromatophores then give one the impression of a spiral or net-work. During further disorganization the chromatophores are as it were crushed to pieces by the growing vacuoles, which pieces are mostly mutually connected only by a narrow (plasm?-) bridge. The cause of this vacuolization of the plasm and degeneration of the chromatophores must be found in the attack of the *Diffflugia*, since the phenomenon always appeared in that half of the cell which was attacked by the *Diffflugia*, from here to spread throughout the cell. In this process the chromatophore would often be found already in an advanced state of disorganization in one half of the cell, whereas the other half of the cell showed no signs yet of vacuolization and the chromatophore in it still seemed completely sound. In one case the first chromatophore was already partly ingested by the *Diffflugia*, when the second showed only a slight shrivelling up, which soon progressed from the middle towards the top of the cell; when the first signs of degeneration began to show at the top a strong vacuolization and aggregation was meanwhile visible already at

the middle. At the same time a stretching of this chromatophore took place, so that it reached over the middle of the cell — possibly it was pushed up — and approached the mouth of the *Diffflugia* (vide fig. 8). The influence radiating from the attacking *Diffflugia* and giving rise to the process is already perceptible a long time before the cell wall is punctured. In one case illustrating this fact particularly strongly a *Closterium* cell was found at 8.10 p.m., which had been attacked by a *Diffflugia* at about  $\frac{1}{3}$  from the end of the cell; then the two chromatophores were still completely sound (Fig. 6 a). At 9.10 p.m. the chromatophore in the cell half, which had been attacked, appeared to recede from the wall, which process proceeded at a slow rate till 10.30 p.m. (Fig. 6 b). Meanwhile, however, the *Diffflugia* appeared to move very slowly towards the middle of the cell. At 7.30 the next morning the *Diffflugia* was exactly before the middle of the cell; in the cell half that had been attacked first the chromatophore was shrivelled up strongly and was aggregated in front of the mouth of the *Diffflugia*; the other chromatophore shows only a slight shrivelling yet (Fig. 6 c). At 1.30 p.m. the cell is emptied and shows in the middle the typical hole. The whole course of the process strongly suggests that the chromatophores are being squeezed out of the cell, maybe as a result of strong internal tension caused by the vacuolization; in this process the degeneration of the chromatophores seems to us a secondary, the plasm vacuolization, on the other hand, the primary phenomenon.

It is worth observing that similar vacuolization phenomena also occur in alga cells during degeneration processes which were not caused by an attack of a *Diffflugia*.

It is thinkable that as a common cause of these phenomena variations of the turgor can be assumed, which in their turn can be determined again by various factors. It also strikes one that in most cases during the emptying action of a *Closterium* cell a bright grey plug of plasm can be observed in the neck of *Diffflugia*, round which the typical orange-red drops will often be grouped archwise (Fig. 9). Probably this plug must be considered as a mass of *Closterium* plasm that has been squeezed out. For, in one case we were in a position to observe that during the emptying process of a cell the plasm of the *Diffflugia* withdrew to within the fundus of the shell, the grey plug mentioned being left behind in the opening of the neck and in contact with the *Closterium* cell. After some minutes the *Diffflugia* plasm lay down again against this neck plug, enclosing it like a hood; for a moment a clear line of demarcation was visible between the two masses of plasm. This phenomenon was repeated a few times, during which, while the *Diffflugia* plasm withdrew, some parts of the grey plasm with elements of the chromatophore were carried along and were transported to the interior of the *Diffflugia* body by a kind of invagination, as in the ingestion by *Amoeba verrucosa*. — For the microphotographs we are greatly indebted to our friend, Mr. Drs. E. C. H. KOLVOORT, Bussum, for the translation into English to Mr. J. B. POLAK, Deventer.



### Samenvatting.

1. *Diffflugia rubescens* Penard voedt zich met den levenden inhoud van *Closterium*-cellen, die opgenomen wordt, nadat de celwand door het dier geopend is.
2. De eigenschappen der door het dier gemaakte opening maken waarschijnlijk, dat het ontstaan der opening een proces van in hoofdzaak mechanischen aard is; een voorafgaande gedeeltelijke oplossing van den celwand langs chemischen weg is mogelijk.
3. Tijdens de opname treedt plasmolyse in de *Closterium*-cel op; daarop volgt een sterke vacuolisatie, gepaard gaande met fragmentatie en aggregatie der chromatophoren.
4. Onverteerde resten der chromatophoren worden in den vorm van roodbruine, bolronde lichamen uitgestooten.
5. Enkysteering staat met de voedselopname niet in onmiddellijk verband.

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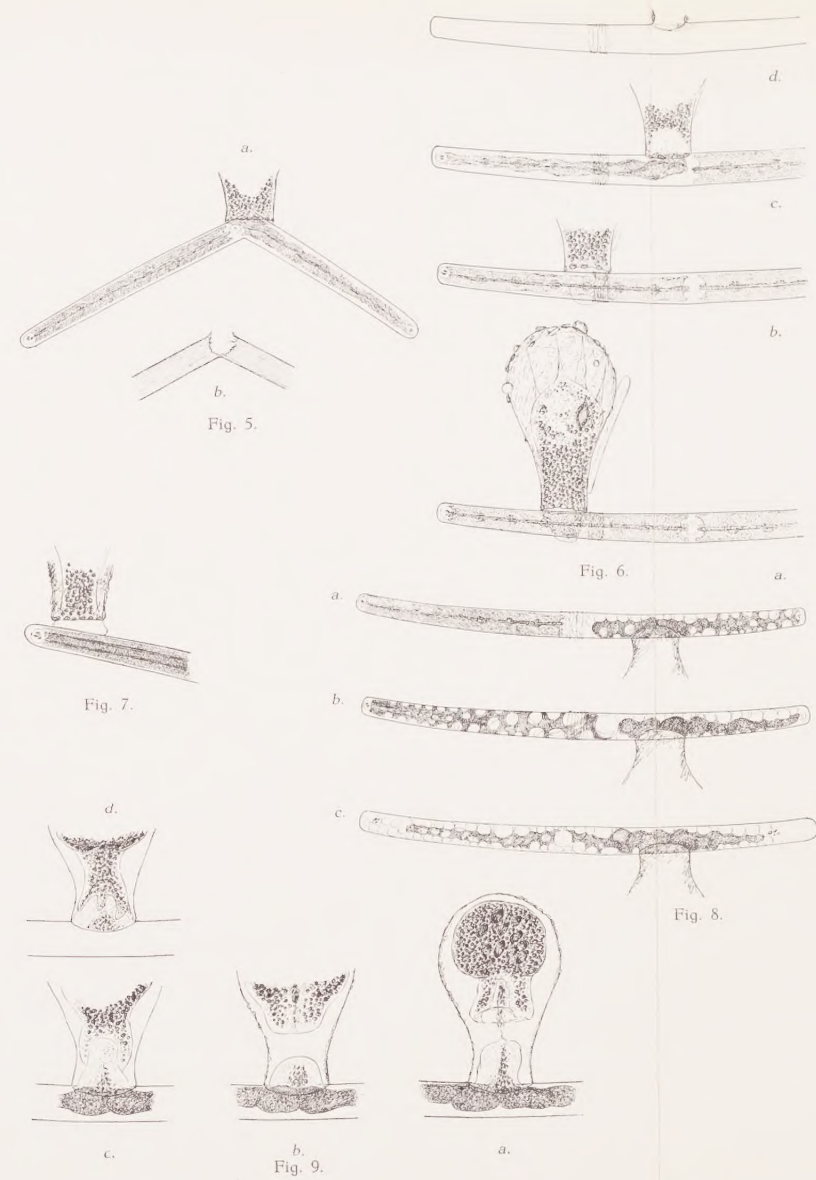
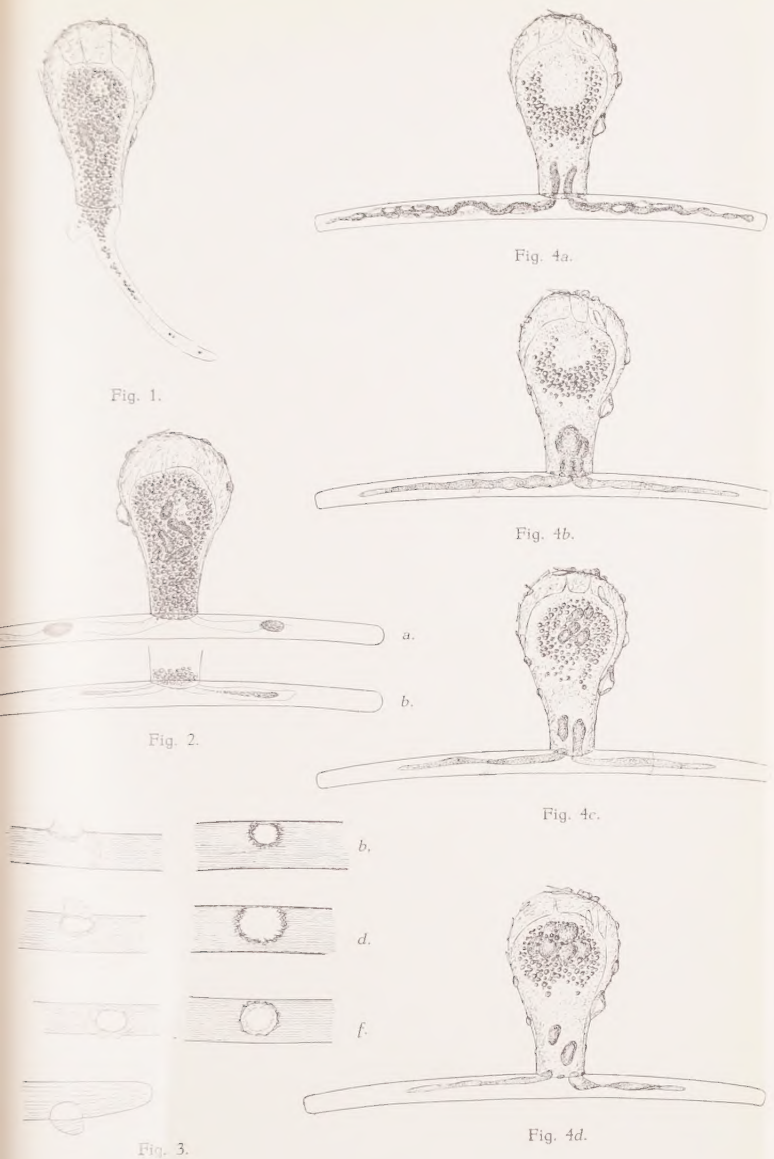


Fig. 1. *Diffflugia rubescens* PENARD. Living, active individual, with pseudopodia, epipodia, drops of pigment and remnants of food.  
Fig. 2. Two stadia of ingestion of the remnants of the chromatophores by the pseudopodia.  
Fig. 3. The holes in the *Closterium* cell wall, a, from aside, the others more or less from above. c. A piece of the cell wall is cut out and turned back. g. (after POISSON and MANGENOT). The hole in the cell wall made by *Vampyrella Closterii*.  
Fig. 4. Four successive stadia in the ingestion process of the contents of a *Closterium* cell; only the two chromatophores are shown.  
Fig. 5. a. *Closterium* cell cracked by the sucking action of *Diffflugia*. b. The same cell after ingestion of the contents, with the hole in the cell wall.  
Fig. 6. Four stadia of the ingestion process. a. First contact. b. The cell held from below by a lump of plasm. c. Ingestion of the chromatophores. d. The hole in the cell wall.  
Fig. 7. First contact of *Diffflugia* with the *Closterium* cell. Pseudopodia drawn in; a lump of plasm touches the cell wall (cf. fig. 11).  
Fig. 8. Disorganization of the *Closterium* chromatophores; strong vacuolization of the *Closterium* plasma.  
Fig. 9. Details of the ingestion process.  
Fig. 10. *Diffflugia rubescens*. Shell in longitudinal optical section.  
Fig. 11. First contact (cf. fig. 7).  
Fig. 12—14. Holes in the *Closterium* cell wall; 12 and 13 from aside, 14 aslant from above.



